PRACTICAL MUSCLE TESTING
AND
THE TREATMENT OF MUSCULAR ATROPHIES.

W. S. HEDLEY, M.D.

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PRACTICAL MUSCLE TESTING

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THE TREATMENT OF MUSCULAR ATROPHIES

BY

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WITH ILLUSTRATIONS

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PREFACE.

THE following pages will fulfil their purpose if they prove of any service to the student who is looking for "more light" on the diagnosis and treatment of a class of cases which rank amongst them morbid conditions the most obscure and intractable that are known to medicine. In dealing with Progressive Muscular Atrophy it is idle to talk of "cure"; but it is not unreasonable to affirm that retardation and even arrest are amongst the possibilities of nature and of art. The latter will avail itself of those agents which can exercise a demonstrable influence on general nutrition. Amongst these, Electricity holds an important place;—not the traditional electricity which trusts alone to localised applications, but those modern procedures of general electrisation, the most effective forms of which are probably to be found in the sinusoidal water-bath, and in currents of great frequency and high potential. It is fully proved by exact physiological experiment that the latter have a remarkable influence on the nutritive exchanges and on cell life. It is to be remembered that the best that treatment can accomplish is so to modify the perverted vital activities of cells and tissues as to strengthen their resistance to morbific agencies, and bring into play their normal recuperative power. "Cure" is the work of the living cell.

In trying to make a useful compilation the writer freely avails himself of the works of recognised authorities, and this in each individual instance he does his best to acknowledge.

8 Mansfield Street, W.
July, 1897.

CONTENTS.

I.—PRACTICAL MUSCLE TESTING.

CHAPTER I.

PAGE
Introduction 3

CHAPTER II.

Laws of Normal Electro-Muscular Contractility 7

CHAPTER III.

Explanatory 15

CHAPTER IV.

Some Sources of Error 19

CHAPTER V.

Laws of Abnormal Contraction 22

CHAPTER VI.

Partial Reaction of Degeneration 30

CHAPTER VII.

Value of Reaction of Degeneration in Diagnosis 34

CHAPTER VIII.

The Motor Points 39

CHAPTER IX.

PAGE
ELECTRO-CUTANEOUS SENSIBILITY . . . 54

CHAPTER X.

THE ACTION OF MUSCLES AND THE CONSEQENCES OF THEIR PARALYSIS 65

CHAPTER XI.

DEFINITIONS, EXPLANATIONS AND MEMORANDA . 77

II.—THE TREATMENT OF MUSCULAR ATROPHIES.

CHAPTER I.

CLASSIFICATION OF MUSCULAR ATROPHIES . . 89

CHAPTER II.

TREATMENT OF MUSCULAR ATROPHIES, MYELOPATHIC 96

CHAPTER III.

TREATMENT OF MUSCULAR ATROPHIES, MYOPATHIC 108

CHAPTER IV.

TREATMENT OF MUSCULAR ATROPHIES, NEUROPATHIC 109

CHAPTER V.

THERAPEUTIC EXERCISES AND OTHER FORMS OF TREATMENT 116

I.

PRACTICAL MUSCLE-TESTING.

PRACTICAL MUSCLE-TESTING

CHAPTER I.

INTRODUCTION.

FOR information about the motor nervous system we interrogate the muscles. The nervous apparatus does not for the most part easily lend itself to direct and independent observation. But so far as its integrity is necessary for normal muscular motility, it is evident that disturbance in the latter, may become an outward sign of an inward nervous lesion. It will be suggested that a great exception to this presents itself in those progressive muscular atrophies named "myopathic," in which so far as is known, the nervous system is intact, and the condition a primary degeneration of muscular fibre. But even here the negative results of electrical exploration afford useful evidence by exclusion.

For the clinical appreciation of pathological changes in living muscle various forms of stimuli are brought to bear upon its contractility, and comparison is made with corresponding manifestations in health. As a starting point therefore in testing the condition of a muscle it is necessary first to be familiar with its normal behaviour under stimulation, as well as to recall some points in connection with the mechanism of its action and the consequent results of its paralysis. It may be convenient first to consider the *electrical* stimulus in health and in disease.

It is a question whether, in the "already over-crowded medical curriculum," room can be found for electro-therapeutics. But at least it may be conceded that a useful, and even a necessary, part of every medical education is the acquirement of a certain familiarity with the technique of Electro-Diagnosis. To attempt any adequate clinical study of diseases of the nervous or muscular systems without this aid, is to grope about in the uncertain twilight for something that might be looked for in the light of day. Uncontrolled by the patient's will, uninfluenced by his story, the electrical reactions of a muscle tell their own tale, and often reveal the one objective feature, the one distinctive physical sign that alone is necessary to differentiate the morbid change. It is scarcely an exaggeration to say that what the stethoscope is to pulmonary and cardiac conditions, what urine analysis is to renal disease, such is electrical exploration to normal and abnormal conditions of the neuro-muscular system. By this means not only is it often possible to distinguish with certainty disease of the lower segment of the motor path, to affirm the presence of a neuritis, or of an anterior polio-myelitis, to exclude the paralysis of cerebral disease, of diseases limited to the white matter of the cord, of hysterical paralysis, of pure myopathies, but there are cases frequently occurring where the distribution among the muscles of the abnormal electrical reactions may lead to a localisation yet more exact, and disclose with something like certainty the actual seat of the morbid process.

It must not be imagined that an electrical diagnosis is quite an easy and simple matter. It requires a considerable experience and involves a procedure that ought to be rigorously exact, approaching, indeed, as far as possible the conditions of a physiological experiment. Yet there is nothing mysterious in the methods nor complex in the instrumentation. The merest outline only of electrical knowledge is necessary. An induction coil and a battery of 30 or 40 cells is the only apparatus required. The so-called "Combined Battery" is of various forms, and all of these are fully explained and illustrated in the catalogues of the instrument makers. There should be a double "current-collector," *i.e.*, a means of throwing the cells into action one by one, commencing with any particular cell that may be desired.* The induction coil should be of the "sledge" type, *i.e.*, one in which the secondary coil can be pulled over the primary. It is of great consequence that the contact-breaker work evenly and at the same note, and that its rapidity of vibration be easily adjustable. A milliampère meter is a *sine quâ non,* and there must usually be a means of securing for it a good level by screw adjustment or otherwise. The battery must also be provided with a "current changer"† and a "pole changer"‡.

To clear the ground it may here be called to mind that there are used in medicine at the present day three chief types of electrical discharge ("current"). These are distinguished by the character *i.e.*, by the frequency, magnitude and wave-type of the electromotive force that produces them. This electromotive force may be:—

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* Or in place of this a Rheostat, by means of which resistance is gradually diminished to the passage of a constant electromotive force.

† For changing quickly from Galvanic to Faradic.

‡ For changing the sign (+ or −) of the testing electrode.

I. Continuous.	(*a*) Produced by a primary or secondary cell ("galvanic"). (*b*) Produced by certain continuous-current dynamos.
II. Alternating.	(*a*) Dissymmetrical (medical induction coil, "Faradic"). (*b*) Symmetrical (alternating current dynamos).
III. Static and kindred forms.	Influence machines and other high frequency apparatus.—The discharge being convective (static breeze), conductive, or disruptive, the latter oscillating (static induced), or non-oscillating ("Franklinic"), currents.

In electro-therapeutics it is necessary to understand and handle all of these; but for the present purpose only two of them need be considered, viz., (1) that form of continuous current produced by the electromotive force of a primary or secondary cell ("Galvanic") and (2) that form of alternating current produced by the electromotive force of the medical induction coil ("Faradic"). In other words as already stated the ordinary "Combined Battery" with its two currents is sufficient for the purpose of electro-diagnosis; although it is probable that as more modern currents come to be better understood, their physical character will be found to give them diagnostic value.

CHAPTER II.

THE LAWS OF NORMAL ELECTRO-MUSCULAR CONTRACTILITY.

IF a continuous current be applied to an uninjured nerve or muscle in the living body, the extent to which that nerve or muscle is excited will vary *(a)* according to the pole which is applied; *(b)* according to whether the circuit is opened or closed; *(c)* according to the intensity used. And inasmuch as this current acts as a stimulus at the moment that the circuit is closed (closure contraction), and also at the moment it is opened (opening contraction*), and as this occurs at both poles, it is evident that the neuro-muscular apparatus may be excited in four different ways. In other words there are to be systematically studied four "electrical reactions," viz., one at closure and one at opening with the negative pole applied to the part under observation, and one at closure and one at opening with the positive pole applied.

TECHNIQUE OF ELECTRICAL EXPLORATION.

CONTINUOUS CURRENT.

Now selecting a normal "subject" (the experimenter will do well to begin by trying upon himself) let the large or indifferent electrode (having a superficial area of say

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* This is only to be accepted as a broad general statement. There are effects know as "Duration" contraction during the steady flow of a strong current, and perhaps during the steady flow, even of ordinary currents there is shortening of the muscle.

100 sq. cm.) be well wetted in hot water, attached to the positive pole (anode), and placed upon the sternum or abdomen, or most conveniently between the shoulders. It may be kept in position by means of a towel crumpled up and pushed in between the electrode and the clothing. Taking now the "small" "active" or "testing" electrode (having a diameter of say 1·5 cm.), let it be well wetted and attached to the negative pole (kathode). Then holding this electrode by its interrupting handle (the finger upon the interrupter) place the electrode upon a motor point. Suppose the latter to be that of the ulnar nerve at the elbow, about an inch above the condyle, at the inner border of the triceps.* Now with one hand holding the electrode upon this point, with the other hand move the handle of the current collector until an electromotive force of 6 or 8 volts (5 or 6 cells) is secured. Continue to put more cells into action, and as the electromotive force is gradually increased, *i.e.*, as fresh cells are added, keep closing the circuit at short intervals by means of the finger upon the interrupter in the handle of the electrode. At one of these closures a muscular contraction will appear—a "short sharp well-defined jerk." Now keeping the circuit closed, note the intensity of the current as registered by the galvanometer. It may be one milliampère.† In this case the testing electrode being attached to the kathode, and the contraction having been noted at the closure of the circuit, it is therefore a "kathodic closure contraction" (K.C.C.), and the cur-

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* Any one making the tests upon himself will, for convenience, select some part of the body other than the arm.

† The intensity required to produce contraction will vary with the stoutness and muscular development of the individual and other circumstances affecting current density.

rent necessary to produce it being 1 ma. (in this particular instance) the fact is expressed K.C.C. = 1 ma. Now the circuit being kept broken by means of the interrupter, but with the testing electrode still upon the same motor point, move the switch upon the element board, which has hitherto been pointing to normal (N), across to the reverse side (R). The testing electrode which was before kathode now becomes anode. Put back the handle of the cell collector to zero and gradually throw into circuit one cell after another as before. Still keep making occasional closures by means of the interrupter. A moment will come when another contraction occurs—"anodic closure contraction" (A.C.C.). Keeping the circuit closed, again read the galvanometer. It perhaps now may register 2 ma., therefore A.C.C. = 2 ma. But only one milliampère was required to produce the "kathodic closure contraction"; therefore kathodic closure contraction is a stronger stimulus than anodic closure contraction, and the fact is noted K.C.C. > A.C.C. With the electrode on the same motor point, but this time keeping the circuit closed, increase the intensity as before, at the same time occasionally breaking circuit by means of the interrupter. Another contraction soon becomes visible. Again read the galvanometer—perhaps it marks 3 ma. Here the testing electrode being anode and the contraction occurring at opening or break of the circuit, it is an "anodic opening contraction;" and the strength of the current necessary to produce it being 3 ma., A.O.C. = 3 ma. But anodic closure contraction required only 2 ma. Therefore the anodic closure contraction is (in this case) a stronger stimulus than the anodic opening contraction, *i.e.*, A.C.C. >

A.O.C. (In the case of some nerves A.O.C. appears before A.C.C. Sometimes these two stimuli are of equal strength. The cause of the difference is a physical one and depends perhaps on polarisation currents, as well as on relative differences in current density, the latter in its turn depending on differences in the nature and bulk of the structures through which the current has to pass on its way to the nerve). Again, having broken circuit but with the testing electrode on the same motor point put the reversing switch back to normal (marked N. on the element board). This makes the testing electrode once more kathode. Again putting the handle of the cell collector back to zero gradually increase the intensity of the current, and when this has attained a considerable amount, say two, three, or four times the strength of the current in the preceding test, break circuit by means of the interrupter. Perhaps a contraction follows. The galvanometer may mark 12 ma. Here the testing electrode being kathode, and the observation made on opening the circuit, it is a "kathodic opening contraction" (K.O.C.), and as 12 ma. is required to produce it, the fact is noted K.O.C. = 12 ma. If no contraction appear on opening such a circuit, the latter must not be closed again until the cell collector has been put back to zero, on account of the violent closure contraction that would otherwise ensue. Starting again from zero the current is gradually raised to a higher intensity than before and the experiment of opening the circuit repeated. But in actual practice this process ought not to go on indefinitely. In every electrical examination there is a limit beyond which the intensity of a current should not be pushed. With an electrode so small as that used in electrical exploration, not only does the process become insupportably painful as an intensity of 20 to 25 ma. is approached, but no good purpose is served by it, inasmuch as the violent contraction of neighbouring muscles is apt to mask, and may be mistaken for, the contraction of the muscle under examination.*

COIL CURRENTS.

Passing to the induction coil current ("Faradism") proceed experimentally as before. Having attached the large electrode to the positive pole† (the terminal marked + on the element board) apply it to an indifferent part of the body. The small, or testing electrode being connected to the negative pole, the Faradic switches (F) having been turned on, and the contact breaker of the coil having been put to its slowest rate of vibration (its lowest note), the testing electrode held by its interrupting handle is placed over the motor point of the nerve as before. With the other hand placed upon the secondary winding of the induction coil (the secondary having, to begin with, been quite pulled back from the primary) the secondary is gradually pushed over the primary. Having reached a certain point a muscular contraction appears on closing the circuit, quick and lightening-like as in the case of the galvanic stimulus. The figure on the graduated scale which marks the relative position to each other of the primary and secondary coils is now noted.‡

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* The contractions produced in electrical testing ought to be "minimal," and as far as possible ought not to exercise neighbouring muscles.

† Of the secondary wire.

‡ The notation on this scale does not afford any definite measurement of

Now inasmuch as the Faradic current is an alternating current (a given terminal being at one moment negative at another positive) it is obvious that the nerve or muscle subjected to its influence must find itself at a constantly varying potential. The excitation will not, therefore, appear to be of short duration as with the continuous current, which once closed has a fixed potential, and only acts as an excitant at the moment of closure and of opening.* In the case of the induction coil, as the circuit is being constantly opened and closed automatically by means of the vibrator, and as every make and break begets a new current, the excitation is kept up during the whole time the apparatus is in action. Considering that the polarity is constantly changing, the utility of having the terminals marked + and — is sometimes questioned. In this connection it is necessary again to call to mind the fact that, in the secondary circuit of an induction coil the wave type is dissymmetrical, the wave at break being greater in amplitude although more brief in duration than the wave at make, yet the *quantity* of electricity is the same. Or stating the same fact in another way, the same quantity of electricity passes, but it passes in a shorter time at break than at make. In other words the intensity is greater at break than at make. What follows from this? The excitation of a muscle is "a function of the intensity of a current," but intensity is greater at

the actual strength of the current, and the figures are not applicable to other coils; but afterwards in exploring abnormal conditions it may be more or less useful as a standard of comparison to guide the same experimenter, using the same coil, actuated by the same electromotive force.

* Duration contraction may for present purposes be disregarded.

break (the same quantity of current passing in a shorter time) therefore break is the more effective stimulus. In other words the current in question is dissymmetrical, not only in physical character, but (in consequence of this) dissymmetrical also in its physiological effects. It is in view of the preponderating influence of the current at break that it has become customary with instrument makers to assign to an alternating current that direction in which its intensity and physiological effects are greatest. Thus the signs plus and minus on the terminals of the secondary coil have reference to the direction of the current at opening of the circuit.

Reviewing the ground that has already been traversed, it will be seen that the following points have been successively established:—

1. That the normal muscle responds to electrical excitation whether Galvanic or Faradic by a short sharp contraction.

2. That this occurs whether the muscle be stimulated "indirectly," that is to say through its nerve-trunk, or "directly," *i.e.*, by applying the stimulus to the muscle itself.

3. That the Faradic current is an alternating current and acts as a stimulus at both poles.

4. And that besides being alternating it is also dissymmetrical inasmuch as its intensity is greater at opening than at closing of the circuit; and therefore it is that opening is the more effective stimulus. But when opening and closing are by means of a vibrator effected automatically and in quick succession, as in the usual conditions, it is the net result that appears, and opening and closing effects are not distinguishable as such.

5. But it had previously been shown that this is far

otherwise with the continuous current. Here it appeared that the nerve could be excited in four different ways and to a varying extent. Thus the kathodic closure contraction (K.C.C.) is the strongest stimulus, next comes anodic closing contraction (A.C.C.), or sometimes anodic opening contraction (A.O.C.), and last, and long behind the others, comes kathodic opening contraction (K.O.C.), which is by far the weakest stimulus.

This law may be formulated thus :—

$$\text{K.C.C.} > \text{A.C.C.} \left\{ \begin{matrix} > \\ =^{*} \\ <^{*} \end{matrix} \right\} \text{A.O.C.} > \text{K.O.C.}$$

or graphically expressed thus (after Hoffmann) :—

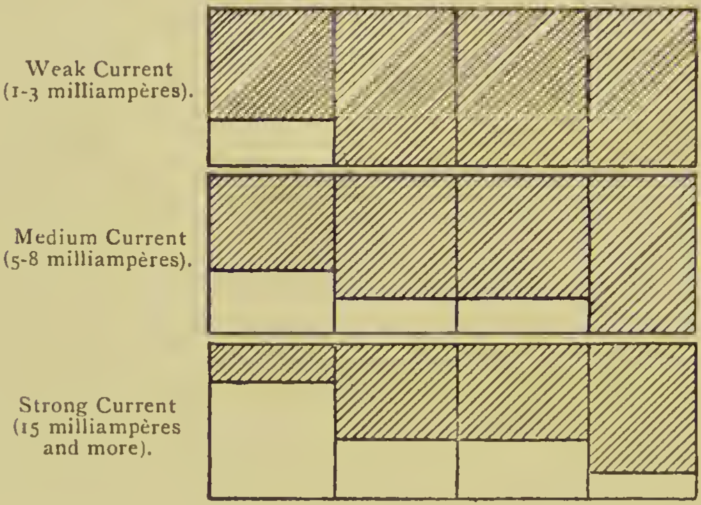

Fig. 1.

where the non-shaded parts represent the relative strengths of the muscular contraction.

Such is the "normal law of contraction," the "normal polar formula"; and it is to this law that the Physician must look for light and leading in his electrical diagnosis —for a guiding landmark in his electro-therapeutics.

* The ratio between A.C.C. and A.O.C. not always being the same even in normal muscles.

CHAPTER III.

EXPLANATORY.

HAVING observed the facts, consider something of the rationale of the procedure that elicits them.

ELECTRODES.

Two electrodes have been spoken of,—the "large" or "indifferent," and the "small" or "active" electrode, the latter being applied to the part to be tested, the former to some remote part of the body. Why? The foregoing tests have shown that the excito-motor power of the two poles of the galvanic current are not equal—that they have to be studied separately. It is evident that if both poles are placed on a given nerve or muscle, it is difficult to distinguish the action of one pole from that of the other. The effects of one will mask those of the other. Yet to complete the circuit both poles must be on the body. This difficulty is got round by using what is termed:—

THE UNIPOLAR METHOD OF EXCITATION.

This consists in so arranging that the effects of the "active" or "testing" electrode are strongly marked and clearly seen, whilst those of the other, the "indifferent" electrode, are as far as possible effaced. Therefore the former is placed over the part to be examined and is so small in area that the density of the current is great and its effects strongly marked, whilst the action of the other electrode, both in point of painfulness and motor effect, is minimised by large surface area, and a position far away from the vicinity of large motor nerve-trunks. In other words the indifferent electrode carries a widely diffused current, a current of little "density."

DENSITY.

It must be clearly understood that the *density* of an electric current is a most important factor in the production of physiological effects, a factor scarcely less important than *intensity*, and these two must be carefully distinguished. The "intensity" of a current is that current's strength as registered by the galvanometer; "density" may be described as the ratio between the "intensity" (current strength) and the superficial area of the electrode which applies the current to the body. Suppose an electrode whose surface area is 10 square centimetres and (to complete the circuit) another electrode whose surface area is 1 square centimetre.—Let a current of 10 milliampères pass. It is obvious that as the current at one pole is spread over 10 sq. cm. and at the other only over 1 sq. cm., the large electrode will carry 1 ma. to the sq. cm., the other will carry 10 ma. to the sq. cm., that is—the "current-density" at the small electrode is in such a case, ten times as great as that at the large electrode, and the physical and physiological effects must be correspondingly different. Therefore to compare results in electrical exploration it is necessary always to use the same sized electrodes. In order to express this relationship between the density and intensity of a current a fraction is sometimes used, the numerator of which is the intensity (in milliampères), and the denominator the area of the electrode (in sq. cm.). Thus the fraction $\frac{1}{10}$ would signify that 1 ma. is spread over an area of 10 sq. cm. In other words there is one-tenth of a milliampère to the sq. centimetre.

DIRECTION OF CURRENT.

Questions are sometimes asked especially by those fresh from their physiological studies—as to the influence of *direction* in electrical currents ; a current being spoken of as "ascending" when its positive pole is the nearer to the periphery and the negative the nearer to the nervous centre, and "descending" when the negative pole is the peripheral pole. In practical electro-therapeutics it is best to get rid of any such distinction. To attribute to a current a sedative or an excitant action according to whether it is "flowing in the direction of the nervous impulse or against it" may be regarded as an electro-therapeutic superstition. The phenomena of electrotonus are of course fully recognised but they are to be explained as "polar" not as "directional" effects. It is to be remembered that the conception of "current" and "flowing" are purely conventional ideas. But the difference in action of the two poles is a fact the reality of which cannot be questioned. Whatever is taking place in the interpolar space may be left out of consideration in electro-diagnosis and the effects may be regarded as purely polar.*

Motor Points.

It is long ago since Duchenne of Boulogne recognised the fact that there are certain well-defined points of the body where stimulation of the nerve or muscle is especially effective. A familiarity with these motor points is one of the first requirements in electrical exploration. The experimenter, trying as usual on himself, will soon be struck by the well-defined character of these points, inasmuch as an astonishingly small movement of the electrode materially alters the result. (See Chap. VII., and Diagrams).

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* A little consideration will show that in experimenting on the living body it is practically impossible to secure by the unipolar method that a current pass through a nerve in only one direction. And not only this, if the point be submitted to a simple experiment it can easily be shown that the position of the large electrode, whether above or below the small one, does not make any difference in the result.

CHAPTER IV.

SOME SOURCES OF ERROR.

BODY RESISTANCE.

MANY pitfalls lie in the path of the electrical explorer :—

(1). In testing with the induction coil current, attention has been drawn to the necessity for being sure, when making comparisons of excitability on symmetrical parts, that the "*Resistance*" of the two sides of the body is the same. With the continuous current an exact measurement can be made without considering the Resistance of the circuit ; and therefore since the introduction of the galvanometer, the question of body resistance has much diminished in importance. But no instrument is as yet easily available for measuring the current of the medical induction coil. In order therefore to compare the relative excitability of corresponding structures on each side of the body by means of faradism, it is not sufficient to apply a current of a magnitude denoted by the distance apart of the primary and secondary of the coil. The figure on this scale might be the same in both cases, yet the strength of the current might be different on account of difference in the relative resistance of the two sides of the body. In other words, effects might be put down to increase or diminution of excitability which are really due to increase or diminution of the strength of the stimulus.*

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* The relative resistance of two symmetrical points can be roughly tested by taking a certain definite number of cells, and noting the intensity of the

ELECTROTONUS.

(2). The excitability of a nerve to one pole may be modified by the fact that that nerve may just previously have been under the influence of the other pole. This source of fallacy is avoided, as far as possible by keeping the current broken except at the actual moment of testing. In testing opening contraction this cannot of course be done, therefore the current must be allowed to pass for a *given* time in each case so that any modification of excitability will at least have been looked for under the same experimental conditions.*

AVOIDANCE OF STRONG CURRENTS.

(3). It has already been stated that the use of strong currents in electro-diagnosis, besides being a cause of needless pain, is apt to be misleading. A strong stimulus does not limit its action to the part covered by the electrode, but diffuses widely to other, perhaps sound groups of muscles, the contraction of which may mask the action of the muscles immediately under observation. It is, therefore, necessary when currents of any consider-

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current which passes through the body. Then taking the same cells and the same electrodes equally well wetted and applied exactly to the same part on the other side, for the same length of time, note the intensity and compare it with the side first tested. The greater current points to the lesser resistance.

* With reference to this point it is necessary that the instrumentation be scrupulously the same where results have to be compared. It has been pointed out by Rouxeau and others that the polarisation current may play a very important part in the production of opening contractions.

able strength are used not only to carefully watch, or feel with the finger, the contractions of the muscle under examination, but as far as possible to put out of action (by fixation) muscles not under observation that would otherwise answer to the stimulus.

MIXED EFFECTS.

(4). Another difficulty presents itself in more or less chronic cases. When the muscles bit by bit become involved in the process of degeneration it is difficult to distinguish the behaviour of the unsound fibres from that of the fibres not yet affected. Here although there may be apparently no inversion of the formula it may sometimes be noticed that the quick sharp normal contraction may be followed by one of a typically " sluggish " or lazy character. It is very necessary to keep this point in view. Its importance in testing, for example, a case of progressive muscular atrophy is evident.

THE REACTION OF EXHAUSTION.

(5). Because at first a muscle responds to faradic stimulation by a contraction apparently normal, it must not be always concluded that therefore the condition of its fibres is unimpaired. There are cases where it will be observed that if the stimulation be repeated a few times in succession, there will eventually be no response. The excitability of the muscular fibre has become quickly exhausted—a fact which points to some morbid change. It is the " reaction of exhaustion."

CHAPTER V.

The Laws of Abnormal Contraction.

The normal law of contraction is the same for nerves and muscles, *i.e.*, the muscle answers by a contraction to an electrical stimulus, whether that stimulus be applied *directly* to the muscle, or be applied to it *indirectly*, *i.e.*, through the medium of the nerve trunk.* It is obvious, as already explained, that it cannot be easy to separate the effect of directly stimulating the muscular fibre, and of stimulating the nerve filaments that pass through it. The effects must generally be mixed effects. Not so, however, in certain abnormal conditions. When the function of the nerve is in abeyance it is difficult to obtain more than two reactions, viz., K.C.C. and A.C.C.; and even where the functions of a nerve are only interfered with and not lost, very marked differences may exist between direct excitation of the fibres of a muscle and their excitation through their nerve. Deviations from the standard of normal response to electrical stimulation, point to corresponding deviations from the standard of health, and to definite anatomical changes in the nerves or muscles that display them.

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* Whenever there is an opening contraction, or a duration tetanus readily observable, we may assume an abnormal constitution of the muscular fibre (De Watteville).

QUANTITATIVE CHANGES.

There may be alteration in the *quantity* or extent of the contraction. Such changes are classed as *quantitative* changes, and may consist of increase, diminution, or total loss of excitability. "Excitability" is measured by the minimum intensity required to produce a visible contraction. The excitability of a muscle must be distinguished from the *force* of a muscular contraction. An atrophied muscle may have only a slight *force* of contraction, but a *perceptible* contraction may be produced by the same strength of current that will produce a contraction in the corresponding muscle of the sound side. The excitability of the two muscles, therefore, is the same, but the force of the contraction is different. Thus a lowering of excitability in a muscle is not always a measure of the extent of its atrophy.

QUALITATIVE CHANGES.

There may be alteration in the character or *quality* of a contraction. These are known as *qualitative* changes. Thus instead of a short sharp jerk the contraction may be "lazy" and "sluggish," or there may be changes in the "normal polar formula."

Having become familiar with the latter, and acquired some knowledge of the topography of the motor points,* as well as inquired into the rationale of the procedure followed in electrical exploration, a standpoint has been gained from which to apply this knowledge to the uses of clinical diagnosis.

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* Page 38, *et seq.*

EXCITABILITY.

There is no way of expressing in any absolute terms the "normal excitability" of nerve and muscle. In different persons even in health there are variations in the "quantity" or extent of the response to electrical stimulation; and the reason of this is largely a question of electro-physics. The fact that the nerve or muscle may be at varying depths from the surface in different individuals, or surrounded by structures of greater or less electrical resistance, would influence the density (and therefore physiological effect) of the current that falls upon a particular nerve or muscle. In point of fact the clinical importance of excitability is apt to be over-rated on account of the practical difficulty of estimating it under precisely similar experimental conditions. In examining a nerve or muscle on one side of the body supposed to be diseased, advantage may be taken of the sound side to compare it with. If both sides are affected, experience in like cases must be the guide; or a comparison must be made with the corresponding structures of a sound limb; or the behaviour under electrical stimulation of a suspected nerve may be compared with that of other nerves known to present similar reactions. Erb says that as a matter of experience it is found that superficial nerves exhibit the first K.C.C with an intensity 0·5 to 2 ma. and from 0·05 to 0·2 absolute density. In order to eliminate the necessity of comparison with other individuals and to get a standard of comparison upon the same person, Erb suggests that 4 pairs of nerves be taken as standards for examination, viz., the facial, spinal accessory, ulnar, and peroneal. It will be found that the relative intensity required to produce muscular contraction through these nerves is for the facial 1·2 ma., spinal accessory ·5, ulnar ·4, peroneal 1·5 This relationship is fairly constant and especially so in the case of the ulnar and peroneal. So that a standard of comparison is thus secured. In other words it can be judged whether the effect of Galvanic and Faradic excitation is increased or diminished *i.e.*, whether the first contraction is obtained with a stronger or a weaker current than normal.

Increase of Excitability.

Increase of excitability to electrical stimulation uncomplicated by other abnormal electrical reactions may be designated "simple" increase. It is not very commonly met with but may occur in the early stages of cerebral disease, in tetanus, in transverse myelitis, in epilepsy, in some forms of hysterical paralysis, in persons under the influence of strychnine, and in other irritative conditions of motor centres.

Diminished Excitability.

Simple diminution of excitability may occur in muscular atrophies (myopathic or consequent on articular disease) and in old standing disease of nervous centres.

Qualitative Changes in Excitability.

In passing from the quantitative to the qualitative alterations in electrical excitability recall once more the normal formula for the reactions of nerve and muscle* :—

$$\text{K.C.C.} > \text{A.C.C.} \left\{ \begin{matrix} > \\ = \\ < \end{matrix} \right\} \text{A.O.C.} > \text{K.O.C.}$$

Whatever the intensity required to produce the contraction these reactions follow each other in regular succession, and the character of the contraction is quick and lightning like—a short sharp jerk. But in certain pathological conditions this is not so, and the A.C.C. appears first, *i.e.*, with a smaller current, than does K.C.C. (inversion of the formula) ; the contraction is lazy or sluggish, and the explorer finds himself in the presence of the "Reaction of Degeneration." This degeneration is characterised by "the diminution and loss of Faradic excitability in both nerve and muscle, whilst the galvanic excitability of the latter remains unimpaired, is sometimes notably increased and undergoes definite qualitative modifications." Of the latter the most characteristic is the "sluggish" contraction, and this appears and disappears slowly, quite unlike the quick "lightning-like" contraction of the normal muscle. This "sluggish" contraction is characteristic and is often alone sufficient to mark the reaction of degeneration. But in a typical case the action of the kathode is no longer the preponderating one. The formula is no longer K.C.C. > A.C.C. The effect of the negative pole only equals or even becomes less than that of the positive pole (K.C.C. = A.C.C. or K.C.C. < A.C.C.).

To follow the course of these changes in their most typical form, consider the case of a nerve cut or bruised.* If the muscles which it innervates be at once examined there is perhaps no very appreciable change;† but soon (in the course of a day or two) the *galvanic* excitability of both nerve and muscle diminishes. At about the end of the first week, however, instead of a diminution, the excitability of the muscle shows a tendency to increase, whilst that of the nerve has continued to diminish or may even be altogether lost. If it has not already disappeared, the excitability of the nerve grows less and less,

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* What has been already stated must here be remembered, viz., that on Faradic stimulation a muscle may at first appear to act normally, but on continuing the stimulation it will be found that exhaustion of the muscle sets in much more rapidly than under normal conditions.

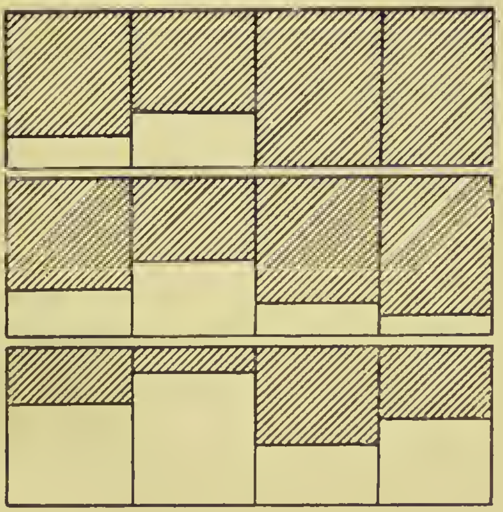

FIG. 2.

and that of the muscle continues to increase until there comes to be a marked difference in their relative excitability. The nerve will probably soon cease to respond,‡ and on direct stimulation of the muscle there is noticed, perhaps between the second and the fourth week, "inversion of the formula." K.C.C. is no longer the stronger stimulus, as is shown in the above diagram (after Hoffman) for comparison with that on p. 14. Another change also has been developing. Instead of a short sharp well defined contraction, the muscle displays a reaction which is "lazy" or "sluggish"—a contraction that may almost be called "vermicular." Now what is the behaviour of nerve and muscle to the current of the *induction coil?* Commencing about 24 hours after the injury there is a steady diminution of excitability (in both structures) going on to complete loss, the latter probably occurring about the end of the second week. Reverting to the Continuous Current:—the condition of exalted "galvanic" excitability lasts for a month or two and then suffers a gradual diminution, while the qualitative change, *i.e.*, the inversion of the formula and the sluggish contraction remain. If the degenerative changes continue to progress, all contractility disappears, the A.C.C. being the last to go, and the muscle ceases to respond to any stimulus, whether galvanic or faradic, direct or indirect. This state of things may be arrived at in weeks or months. If there be arrest and regeneration, returning function first shows itself in an approach to restoration of equilibrium between the action of the affected muscles and their antagonists, and by the effects of gravity ceasing to produce deformity owing to the supporting muscles once more being brought into action. Muscular action in response to electrical stimulation appears later.*

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* As an example take the ulnar nerve injured in the axilla and stimulated at the elbow.

† There may be slight increase of faradic excitability at first.

‡ Often about the end of the second week.

Return of structure and function may occur at various periods depending upon the surgical progress of the divided nerve. Cases have been recorded where sensation and motor power have been restored in seven or eight weeks. More probably it may be many months or even some years. But no case ought to be given up as hopeless until after two or three years and not even then if *any* improvement be in progress (Bowlby).*

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* During the regeneration of a mixed nerve sensibility is restored first, subsequently voluntary motion, and lastly the movements of the muscles when their motor nerves are stimulated directly. (Landois and Stirling).

The reaction of degeneration in its most typical form thus stands clearly out. It consists of four things :—

1. Abolition of faradic contractility in nerve and muscle.
2. Exaggeration of galvanic contractility in muscle.†
3. A contraction which is "sluggish."
4. Inversion of the normal polar formula.

In other words, R.D. is characterised by the fact that whilst the faradic excitability of the nerve and the faradic excitability of the muscle are similarly affected, the galvanic excitability of the muscle suffers in a way peculiarly its own. To faradism there is loss of excitability in the case both of nerve and muscle ; to galvanism there is loss for the nerve, but increased excitability, inversion of the formula and sluggish contraction for the muscle. And of all the stigmata that stamp the true reaction of degeneration, the most typically characteristic and the most constantly present—the one thing necessary—is this "sluggish contraction."

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* "If the recently severed ends of a nerve be placed and maintained in apposition they will unite. It is probable that in a few exceptional cases this union may occur without any preceding degeneration of the distal end . . . . but it is quite certain that this is not usually the case. . . . . The development of this new nerve tissue requires a great length of time and may extend over a period of six to twelve months before material improvement is demonstrable and sometimes requires a year or two for its completion." (Bowlby. F. Treves' "System of Surgery").

† Accompanying the R.D. there is also, appearing later and disappearing sooner, an increased excitability to mechanical stimulation.

CHAPTER VI.

Partial Reaction of Degeneration.

It often happens, indeed it is the rule rather than the exception, that R.D. does not display itself in so clear and typical a way as that just detailed. Between the normal condition and this complete R.D. very many gradations are possible. When such occur they are known as "partial R.D."

(1). The excitability of the nerve may be not altogether lost, indeed only diminished to a very slight extent; yet the changes in galvanic reactions, *i.e.*, the exaggerated excitability, the inversion of the formula and the sluggish contraction are present.

(2). There may be simple diminution of irritability in both nerve and muscle for coil currents, with sluggish contraction for continuous currents, and without inversion of the formula.

Other anomalous forms are the following: —

(*a*). It has been pointed out by Erb that R.D. or partial R D. may occur in muscles which are not paralysed (lead poisoning sometimes).

(*b*). The muscles may respond by a sluggish contraction to stimulation applied through the nerve trunk.

(*c*). The muscle may respond in a similar sluggish manner to induction coil currents applied through the muscle itself.

These deviations are nothing more than the old story, that in clinical work disease is seldom met with in its absolutely typical aspect. In point of fact the Reaction of Degeneration may exhibit every sort of departure from its classic form. But it is none the less necessary to have the perfect type ever before the mind.*

Nothing has so far been said about the *theory* of the normal formula. For detailed information of what is known on this point the text-books must be referred to.† It may be remembered that Pflüger's law is usually explained by the action of three factors:—(1) The increase of excitability in the nerve under the kathode (kathelectrotonic alteration of excitability) and diminution of excitability under the anode (anelectrotonic alteration of excitability). (2) That at make of the current or "closure" the stimulus is kathodic because an increase of excitability is equivalent to a stimulus. At break or "opening" of the current the stimulus is anodic because under the anode excitability is diminished and at opening it suddenly recovers its former level, and this change is equivalent to a sudden increase. But this relative increase is not so effectual as the actual increase at the kathode, therefore kathodic closure is the stronger stimulus. (3) The points at which a current enters a nerve constitute a collective anode, and the points of exit constitute a collective kathode. The greatest density of a current is at the part of the nerve immediately under the electrode (the "polar region"). The least density at the opposite side of the nerve (the "peripolar region"). Applying these facts to the polar formula it is evident that kathodic closure contraction is the first to appear because the kathode closure, the more effective stimulus, falls upon the region of greatest current density, the polar region. With the anode over the part, and closure made, the stimulus is still kathodic but it falls upon the peripolar region, the region of lesser density. In anodic opening, the stimulus is at the anode, the weaker stimulus, but it falls upon the best (most dense) region. In kathodic opening the stimulus is anodic (because it occurs at opening) and it falls upon the worse, the less dense, the peripolar region, therefore the worse stimulus is falling upon the worst region, and therefore kathodic opening is the weakest stimulus.

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* In this connection a phenomenon may be adverted to, described by Ghilarducci and others, and claimed by them to constitute a new form of the R.D.; they term it R.D. "at a distance" and they thus describe it:—It is characterised (*a*) "by contractions which are produced at closure of the circuit, when the two electrodes are applied at a distance from the muscle in such a way that the muscle is comprised in the interpolar space; (*b*) by the absence of electro-excitability or by the presence of R.D. when looked for by the ordinary method. (*c*). It constantly accompanies R.D. but differs from the latter by the predominating action of the negative pole. (*d*). It persists sometimes months and years after the disappearance of contractility as looked for by the oridinary methods. It is, therefore, the last indication of the existence of the muscular fibre. (*e*). So long as this form of R.D. is present there is still hope of recovery of motor power.

† For example, Waller's "Human Physiology," p. 360.

No explanation of the normal polar formula can be quite satisfactory which does not also account for R.D. This the above entirely fails to do. Another explanation has been sought in the theory of Ehrlich. According to the latter all animal functions are excited by want of oxygen. The respiratory centre is certainly so excited. Perhaps this want of oxygen is the *condition* of muscular contraction?* Electrical excitation, like any mechanical stimulus, may produce a consumption of oxygen in the cells of the muscular or nervous system. This "oxygen-hunger" according to the theory in question would manifest itself by the excitation of these cells. But this excitation would be modified by the products of electrolysis at the poles. Thus:—When with a medium current there occurs a kathode closure hydrogen is eliminated at the kathode. This hydrogen quickly enters into combination with the oxygen of the tissues, and this deprivation of oxygen acts as an excitant, which added to the excitant action of the current produces the strongest stimulus. With closure at the anode oxygen is produced at the pole, which acting as a plus to the oxygen already in the tissues, diminishes excitability and therefore tends to cut down the action of the electric stimulus. At anodic opening there is a *relative* diminution of oxygen which is equivalent to a stimulus. At kathodic opening there is a relative increase of oxygen which diminishes the effect of the electrical stimulation. Reduced to a tabular form this theory would read somewhat as follows:—

AT	NATURE OF STIMULUS.	OXYGEN.	
K.C.	Kathodic	Diminution	Stimulant action of current increased by deprivation of O.
A.C.	,,	Increase	Stimulant action of current diminished by increase of O.
A.O.	Anodic	Relative diminution	Stimulant action of current increased by relative diminution of O.
K.O.	,,	Relative increase	Stimulant action of current diminished by relative increase of O.

---

* See "Extract from Clinical Lecture by Pawlinoff." (Sletoff). *Rev. Int. d'Electrotherapie.*

In other words according to this theory electrotonic alterations of excitability are nothing more nor less than the local electrolytic action of the poles.

In R.D. the progressive increase* in galvanic excitability and the diminution of Faradic excitability at about the end of the second week, would be explained by the excitant action of want of oxygen in the muscle whose nerve is degenerated. The degeneration of the muscle being slower than that of the nerve, the reversal of formula would be explained by the fact that a structure in order to be excitable must not be *entirely* deprived of its oxygen. The action of the anode in certain stages of degeneration will supply just sufficient oxygen to allow the electrical stimulus to act. Therefore as degeneration progresses the number of muscular fibres excitable by the anode will be greater than those excitable by the kathode, inasmuch as the former supplements the little oxygen that remains, and so makes the cells amenable to electrical excitation until absolute degeneration has occurred.

If Erlich's theory could be established it would no doubt offer an explanation, more or less satisfactory, of Pflüger's law, the reaction of degeneration and electrotonic alterations in excitability. But wherever an explanation of the latter is to be sought it is certainly less likely to be found in the phrase "modifying action of the current," or in the theory of "molecular orientation," than in those definite electrolytic effects that are known to occur at the poles.

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* The decrease of galvanic excitability in the muscle at first is probably due to the early degeneration of the intra-muscular nerve tissue before the changes, *i.e.*, the deprivation of oxygen has occurred in the muscle; a deprivation which according to this theory is the cause of the increase in excitability.

CHAPTER VII.

THE VALUE OF REACTION OF DEGENERATION IN DIAGNOSIS.

WHAT then does the reaction of degeneration show? Its physical signs are sufficiently obvious, but what is its pathological significance? It points to abnormal conditions of that part of the motor tract, which commencing at the motor ganglion cell terminates at the muscle in the motor end-plate. It means something wrong in the lower segment of the motor path. But it does not take cognizance of morbid conditions of the "upper segment." It means either that there is a break in the line of communication between the muscle and its trophic centre, or that the trophic centre is itself the seat of morbid change.

Thus the reaction of degeneration, partial or complete, may occur after disease or injury, involving structural alterations in the motor nerve trunks, and after disease or injury affecting the ganglion cells in the anterior cornua of the cord, or the corresponding nuclei of origin in the case of the cranial nerves. The diseases, therefore, in which it may be looked for are:—

1. Those muscular atrophies depending on disease of the anterior cornua of the cord:—Progressive muscular atrophy of Aran-Duchenne type and Charcot-Marie types; infantile paralysis; amyotrophic lateral sclerosis; anterior poliomyelitis, acute and chronic; diffuse myelitis

involving anterior cornua ; transverse myelitis at the seat of lesion.

2. In muscular atrophies of neuropathic origin—traumatic, rheumatic, parenchymatous.

3. In toxic and infectious paralyses :—Lead poisoning with muscular atrophy, diphtheritic paralysis, &c.

The more slow the development of the morbid process the more difficult it is to detect the reaction of degeneration. In such cases the cells of the anterior cornua and the muscular fibres are only gradually, and one by one, encroached upon, so that there are often a sufficient number of sound structures left to give normal reactions, and thereby to mask the reaction of degeneration which would otherwise be apparent. And this state of things may go on until the muscular atrophy is so far advanced that the degenerating fibres are incapable of contraction strong enough to appear through the skin.

The limitations of the usefulness of electricity in diagnosis must be clearly recognised. The reaction of degeneration enables a morbid condition to be referred to the "lower segment," and sometimes to be localised much more exactly. It is evident that degenerative change in a nerve trunk will affect those muscles only which that nerve innervates. The distribution of the abnormal reactions will, therefore, point to the nerve affected. If the motor ganglion cells be attacked, the muscles will be affected according to their physiological grouping, rather than according to any anatomical distribution. Thus the occurrence of R.D. in certain groups of "physiologically related" muscles will point to the probable level of the mischief in the cord. Reaction of degeneration does *not* occur in the paralysis of cerebral disease, excepting when the implication of the nucleus of origin or of the nerve trunk of the cranial motor nerves produces R.D. in the muscles which they supply. It does not occur in hysterical paralysis, whether with or without atrophy, nor is it seen in pure myopathics, in the atrophy of disuse, in atrophies consecutive to articular disease, or in diseases limited to the white matter of the cord. Therefore it is evident that one of the chief uses of electro-diagnosis is to exclude a certain number of morbid conditions in which R.D. is never seen. (De Watteville).

Cases illustrative of the foregoing points occur in numbers in every case-book. I extract one (showing a useful "form" of report) almost at random from the electrical records of the London Hospital (see next page).

Prognosis.—Electrical exploration is also useful in prognosis. The principle can be established that in conditions where R.D. may be expected, the more the electrical reactions diverge from the normal type, the longer the lesion will last and the more difficult it will be to cure.

As an illustration of this, the gravity course and duration of "Bell's Paralysis" are thus indicated by Erb. If the electrical reactions continue normal, recovery will occur probably in two or three weeks. If the R.D. be partial (*i.e.*, if excitability to Faradism be not abolished) six weeks to two months; if R.D. be complete three, six, or nine months may be necessary for recovery; or the latter may never take place.

But the occurrence of R.D. is by no means indicative of irreparable mischief. Complete regeneration is sometimes seen, after profound trophic changes, in cases of

REPORT OF ELECTRICAL REACTIONS.

NAME AND AGE.—*F. Pincott.* DATE.—*July 23rd, 1896.*

MUSCLES.	CONTINUOUS CURRENT.		INDUCTION COIL.	ELECTRO-CUTANEOUS SENSIBILITY. (STATE REGION EXAMINED).	REMARKS.
	(1). QUALITY OF CONTRACTION ("NORMAL" OR "SLUGGISH"). (2). FORMULA ("NORMAL" OR HOW ALTERED).	EXCITABILITY.*	MOTOR REACTIONS.*		
Supinator long. R. Ext. c. dig. R. Abd. Pol. l. R. Ext. c. r. l. R. Ext. c. ul. R.	Sluggish contraction A.C.C. >K.C.C.	Diminished	Lost	Diminished back of R. forearm and fingers supplied by Radial; in the proportion (in fore-arm) of (r.) 2 (l.) 5	In railway employ. Ten weeks ago arm fractured by cart-wheel passing over it. Was treated in a country hospital. On removal of splint at end of 3 or 4 weeks could not move wrist Attended that institution 6 or 7 weeks. Was "ordered galvanism, but machine generally out of order." When sent to the London Hospital there was wrist-drop and electrical reactions as in foregoing report. Seeing that the muscles supplied by median and ulnar show unchanged electrical reactions and also the triceps, the injury can be localised in the musc. spiral below the supply of the latter muscle. 5 m.a. continuous current passed transversely through the arm at seat of injury for 5 min. followed by labile application of a "mixed" current to the affected muscles, sufficient to induce moderate contractions. Thrice weekly. *Aug. 20th.*—Voluntary power to some extent regained in supin. long. *Sept. 10th.*—Voluntary power to some extent regained in exten. wrist and fingers. *Sept. 30th.*—Recovery nearly complete. To cease attending.
Abd. m. dig. R. Flex. c. ul. R. Interossei dors. R. Flex. sub. dig. R. Triceps R. Flex. l. pol. R. Flex. c. rad. R.	Normal	Normal	Normal		

* "Normal," "Increased," "Diminished," "Lost."

paralysis dependent on disease of the lower segment. On the other hand it has already been shown that lesions of the gravest kind may exist without the R.D., *e.g.*, in the paralysis of cerebral disease, or in the case of certain intractable spasms or convulsions.

Although practical muscle testing chiefly confines itself to the presence or absence of the reaction of degeneration, there are many refinements of electro-diagnosis above and beyond this. Indeed it is scarcely too much to say that galvanic and faradic contractility are absolutely normal only when motor centres, motor nerves, and the muscles that they innervate, are absolutely sound and intact.

CHAPTER VIII.

THE MOTOR POINTS.

THE topography of the motor points and its ready application to purposes of Diagnosis are best studied by following the anatomical distribution of nerves. That is to say individual nerves are taken and the points ascertained at which electrical stimulation (applied both to the nerve itself and to the muscle) most readily induces a contraction.

To gain a general idea of the strength of current ordinarily to be employed in diagnosis the student will in the first instance experiment upon himself, and commencing with the face he will quickly learn that, in this region at least, the use of *mild* currents is highly desirable. It is scarcely necessary to add that the frequent use of very strong currents in testing diseased conditions is much to be deprecated; on the other hand, the mistake must not be made of supposing that a muscle does not answer to electrical stimulation in cases where as a matter of fact a stronger current would elicit a response.

The following diagrams fairly well represent the motor points, but it is to be remembered that the latter may vary not only in different individuals, but in the same individual on different sides of the body; and further, in as much as electrical exploration has often to deal with pathological conditions which alter the contours of the surface (*e.g.*, Bell's paralysis) it is evident that no diagram nor even photograph of the motor points can be uniformly and exactly applicable in every case. In point of fact it is quite possible to exaggerate the importance of such diagrams. The student of anatomy knows where nerves become superficial, and where therefore they are most easily excitable. He also knows that the motor point of a muscle is the point where the nerve enters. Now that point is usually close to the middle of the muscular fibres in the long direction (Castex).

To record clearly the results of electrical exploration such a " Form " as that given on p. 37 is useful.

REGION OF THE FACIAL N. (*Plate I.*).

n. Facial.

Trunk of nerve best reached by applying the testing electrode behind the lobule of the ear, below the external auditory meatus, and pushing inwards, forwards, and upwards; after the nerve divides, the upper branch (supplying muscles above palpebral aperture) the middle branch (muscles between orbit and mouth) and the lower branch (muscles of lips and chin) may be stimulated at the points shown.

n. Posterior auricular.
m. Frontalis.
m. Corrugator supercilii.
m. Orbicularis palpebrarum.
m. Nasal muscles.
m. Levator labii superioris.

(Not shown).

mm. Zygomatici.

(Impossible to differentiate the action of the two zygomatics).

m. Buccinator.

(At a point not shown, below and external to point for zygomatics).

m. Orbicularis oris.
m. Levator menti.

m. Quadratus menti.
m. Triangularis menti.
m. Platysma myoides.

REGION OF THE FIFTH N. (INFERIOR MAXILLARY BRANCH). (*Plate I.*).

m. Temporal.
m. Masseter.
m. Mylo-hyoid.
(At a point just below Quad. menti not shown in Plate).

REGION OF THE HYPOGLOSSAL N. (*Plate I.*).

n. Hypoglossal.
(Difficult or impossible to excite).
m. Omohyoid.
(Push the electrode well in between the two insertions of the sterno-mastoid).

REGION OF THE SPINAL ACCESSORY N. (*Plate I.*).

n. Spinal accessory.
Crossing post. sup. triangle slightly above its middle.
m. Sterno-cleido-mastoid.
m. Trapezius (" Cucullaris ").

REGION OF THE POSTERIOR BRANCHES OF THE CERVICAL NERVES.

m. Splenius.

REGION OF THE CERVICAL PLEXUS.

m. Trapezius.
The trapezius can be excited not only at the point shown but at points between the vertebral column and posterior border of the scapula for the middle (horizontal) and lower (oblique) portions of the muscle.

n. Phrenic.

(At outer edge of lower part of sterno-mastoid; and between the insertions of that muscle).

Region of the Brachial Plexus.*

"Erb's point" for simultaneous excitation of biceps, brachialis ant., deltoid, supinator longus. About an inch above the clavicle, a little externally to the posterior border of sterno-mastoid immediately in front of transverse process of 6th cervical vertebra.

n. Circumflex.

Can be excited at a point not shown at the lower and outer part of the post. inf. triangle. This produces a contraction of the entire mass of the deltoid.

n. Pectoralis major. (Plate I.).

(Ant. thoracic n.).

n. Serratus magnus.

On the inner wall of the axilla. (Not shown).

m. Deltoid. (Plates II. and III.).

m. Teres minor.

About the middle of axillary border of scapula. (Not shown).

m. Teres major.

At posterior fold of axilla. (Not shown).

m. Latissimus dorsi.

At posterior fold of axilla. (Not shown).

m. Supra-spinatus.

Above the inner end of spine of scapula. (Not shown).

m. Infra-spinatus.

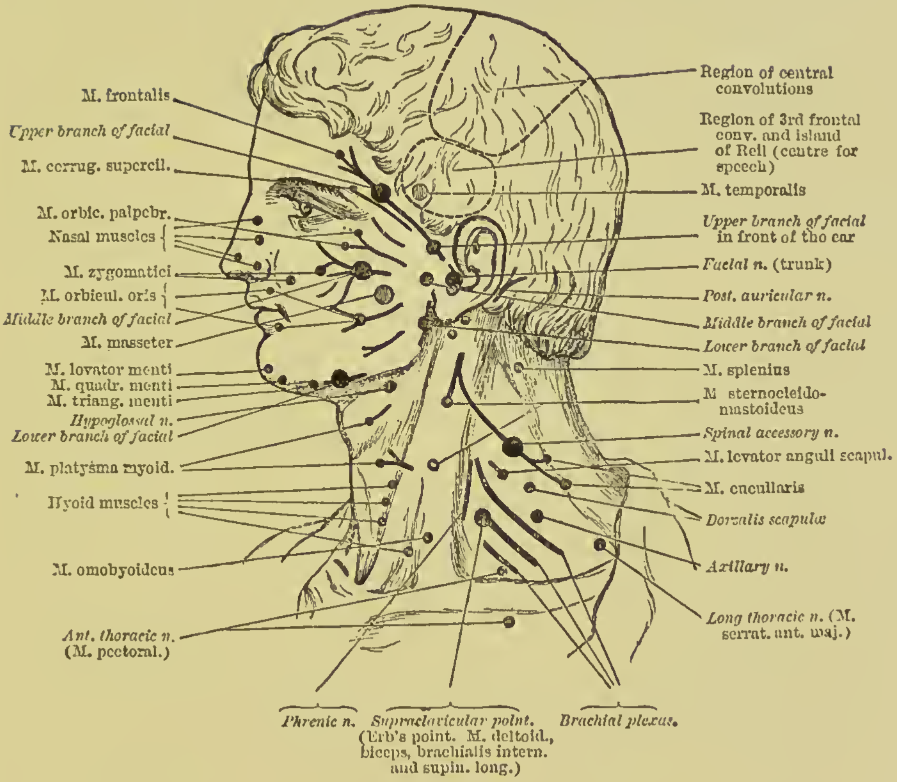
Below the inner end of spine of scapula. (Not shown).

m. Levator anguli scapulæ.

---

* To obtain the best and as far as possible isolated action of muscles, the limb must be relaxed. This is often best effected by the examiner supporting with his left hand the part under examination, so as to suppress all voluntary action on the part of the patient.

PLATE I*



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* Plates I. to XI. are taken with permission from "Medical Electricity," by H. Lewis Jones, M.D. (London: H. K. Lewis).

mm. Rhomboids.

In a depression just below the lower border of the trapezius, when the arm is carried upwards and backwards, a certain number of the fibres of the rhomboid can be thrown into contraction. (Castex). (Not shown).

REGION OF THE THORACIC NERVES. (*Not shown*).

m. Rectus abdominis.

Each m. excitable at two points—one point a little above, the other a little below the umbilicus.

m. External oblique.

At 5 points where it interdigitates with the serratus mag.

REGION OF THE MUSCULO-CUTANEOUS N. (*Plates II. and III.*).

n. Musculo-cutaneous.

(Press testing electrode well under the anterior border of the deltoid and the external border of the coraco-brachialis muscle).

m. Biceps (III.).

m. Coraco-brachialis.

(Just below point for musculo-cutaneous N.).

m. Brachialis ant (II. and III.).

(Press the electrode well in below the biceps avoiding the median nerve at inner part of arm).

REGION OF THE MEDIAN NERVE. (*Plate III.*).

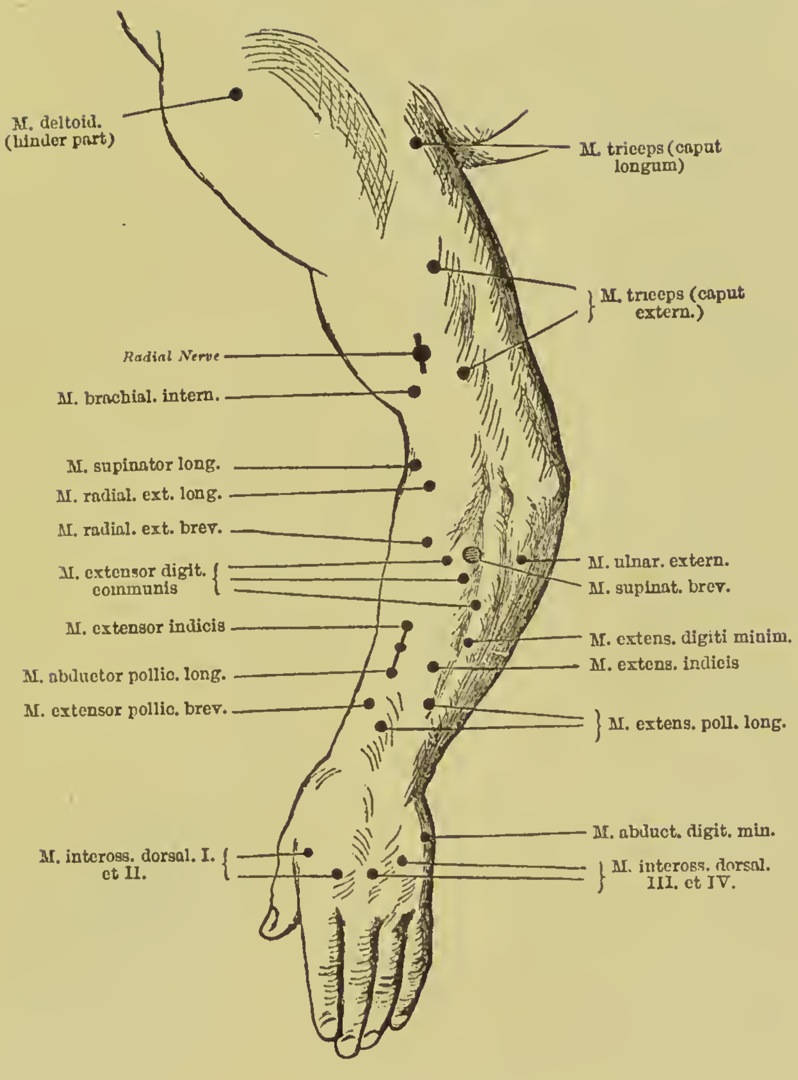
n. Median.

(1. Easily accessible along the whole length of the arm by the inner border of biceps. 2. At the elbow just internal to the biceps tendon. 3. At the wrist between the tendons of the flex. C. radialis and palmaris longus).

m. Pronator radii teres.

m. Flexor c. radialis.

PLATE II.


M. deltoid. (hinder part)
M. triceps (caput longum)
M. triceps (caput extern.)
Radial Nerve
M. brachial. intern.
M. supinator long.
M. radial. ext. long.
M. radial. ext. brev.
M. extensor digit. communis
M. ulnar. extern.
M. supinat. brev.
M. extensor indicis
M. extens. digiti minim.
M. abductor pollic. long.
M. extens. indicis
M. extensor pollic. brev.
M. extens. poll. long.
M. abduct. digit. min.
M. inteross. dorsal. I. et II.
M. inteross. dorsal. III. et IV.

m. Flexor sub. digitorum.

m. Flexor longus pollicis.

m. Flexor brevis pollicis.

Excited at the inner half of thenar eminence by a *feeble* current, otherwise the opponens is also thrown into action.

m. Abductor pollicis brevis.

Excited at the external border of thenar eminence. (Sometimes supplied by radial as well as median). (Roumillac).

m. Opponens pollicis (Plate III.).

(Having found the external border of the first metacarpal bone push the pointed electrode against the anterior surface of that bone (Roumillac). This and the two previous mm. are difficult to excite independently).

m. Lumbricales (2).

REGION OF ULNAR N. (*Plate III.*).

n. Ulnar.

(1. In groove between Internal Condyle and Olecrănon. 2. At wrist on outer side of tendon of flexor c. ulnaris).

m. Flexor carpi ulnaris.

m. Flexor prof. dig.

The point marked is that usually given by the German authors, but it is very questionable whether the deep flexor lying so deeply as it does, can be excited at the front of the forearm at all. The fibres for the ring and little finger can be excited at a point somewhat posterior to the point marked (Castex).

m. Palmaris brevis.

m. Abductor min. digiti.

m. Flexor digiti minimi.

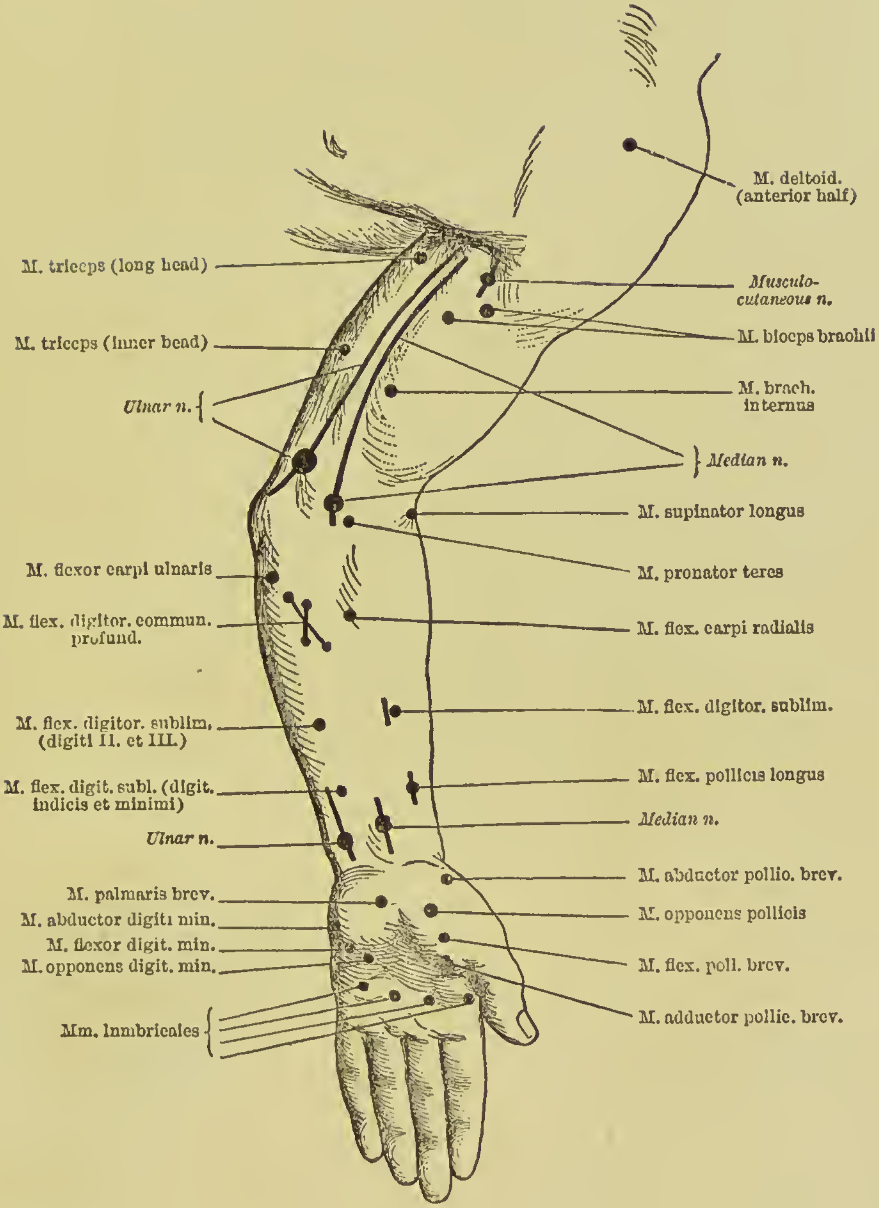
m. Opponens dig. minimi.

m. Adductor pollicis.

mm. Lumbricales (2).

mm. Interossei.

PLATE III.


M. deltoid. (anterior half)
M. triceps (long head)
Musculo-cutaneous n.
M. biceps brachii
M. triceps (inner head)
M. brach. internus
Ulnar n.
Median n.
M. supinator longus
M. pronator teres
M. flexor carpi ulnaris
M. flex. digitor. commun. profund.
M. flex. carpi radialis
M. flex. digitor. sublim.
M. flex. digitor. sublim. (digiti II. et III.)
M. flex. pollicis longus
M. flex. digit. subl. (digit. indicis et minimi)
Median n.
Ulnar n.
M. abductor pollic. brev.
M. palmaris brev.
M. opponens pollicis
M. abductor digiti min.
M. flexor digit. min.
M. flex. poll. brev.
M. opponens digit. min.
M. adductor pollic. brev.
Mm. lumbricales

REGION OF THE MUSCULO-SPIRAL.

n. Musculo-spiral (Plate II.).

(Between triceps and brachialis int. just above the supinator long. "at the point where the n. emerges from the triceps." It will often be found easier to excite this nerve independently by means of a continuous current than by means of an alternating one).

m. Triceps (II. and III.).

m. Supinator longus.

To elicit the action of this muscle well, the examiner should support the arm in the semi-flexed position.

mm. Ext. carpi radialis long. and brev.

m. Supinator brevis.

(The extensor c. digitorum is also excited).

m. Extensor c. digitorum.

m. Extensor indicis.

m. Abductor pollicis longus.

m. Extensor pollicis longus.

m. Extensor pollicis brevis.

m. Extensor minimi digiti.

m. Extensor indicis.

LUMBAR PLEXUS.

REGION OF ANTERIOR CRURAL N. (*Plate IV.*).

n. Anterior crural.

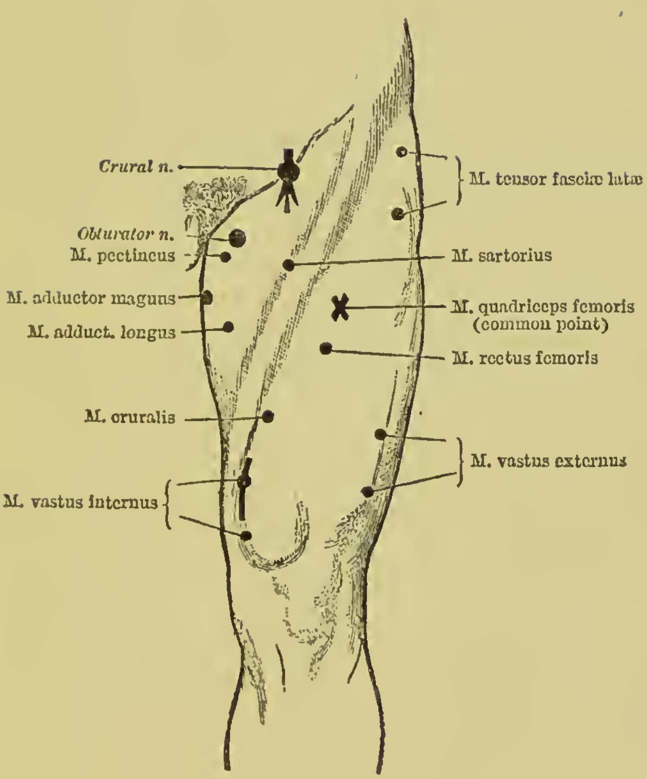
(Motor point in groin just outside femoral artery).

m. Sartorius.

m. Rectus femoris.

(Motor point at the middle of the muscle a little towards the inner border).

PLATE IV.


Crural n.
Obturator n.
M. pectineus
M. adductor magnus
M. adduct. longus
M. cruralis
M. vastus internus
M. tensor fasciæ latæ
M. sartorius
M. quadriceps femoris (common point)
M. rectus femoris
M. vastus externus

m. Vastus internus.

(Motor point, that part of the muscle where it gets free of the rectus and sartorius).

m. Pectineus.

REGION OF THE OBTURATOR N. (*Plate IV.*).

m. Adductor magnus.

m. Adductor longus.

m. Pectineus.

SACRAL PLEXUS.

REGION OF THE SUPERIOR GLUTEAL. (*Plate V.*).

m. Tensor fasciæ fem.

m. Gluteus medius.

(Not shown in plate, but near upper point for G. max.).

REGION OF SMALL SCIATIC.

m. Gluteus maximus.

REGION OF THE GREAT SCIATIC. (*Plates V. and VI.*).

n. Sciatic.

m. Biceps (2).

m. Semi tendinosus.

m. Semi membranosus.

(Difficult to differentiate s. mem. and s. tendin.).

n. External popliteal.

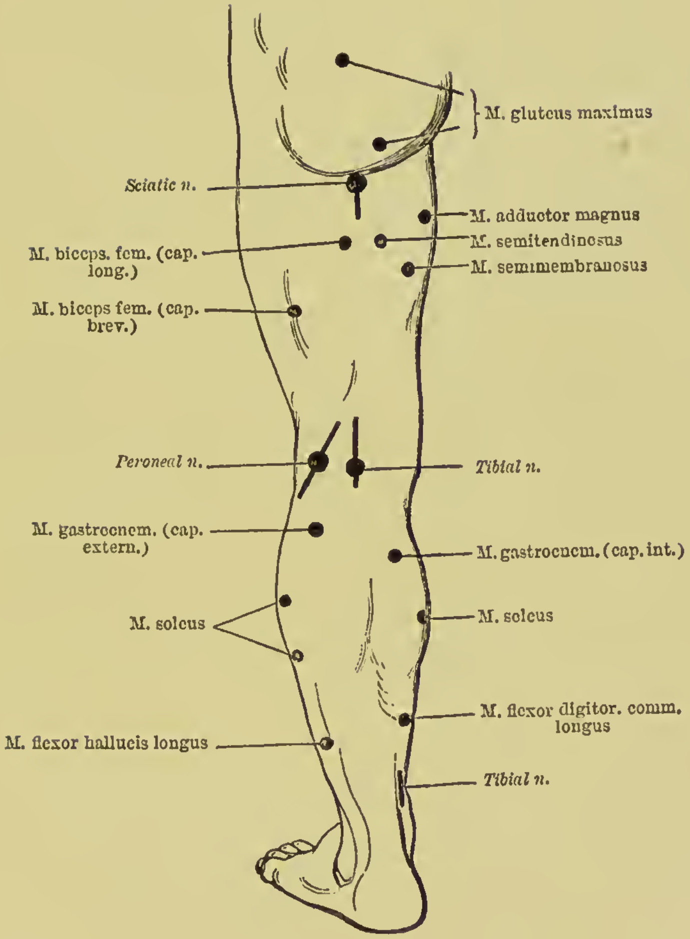
(At outer end of principal transverse fold of popliteal space just above the head of fibula, beside tendon of biceps).

m. Tibialis anticus.

m. Extensor digitorum communis.

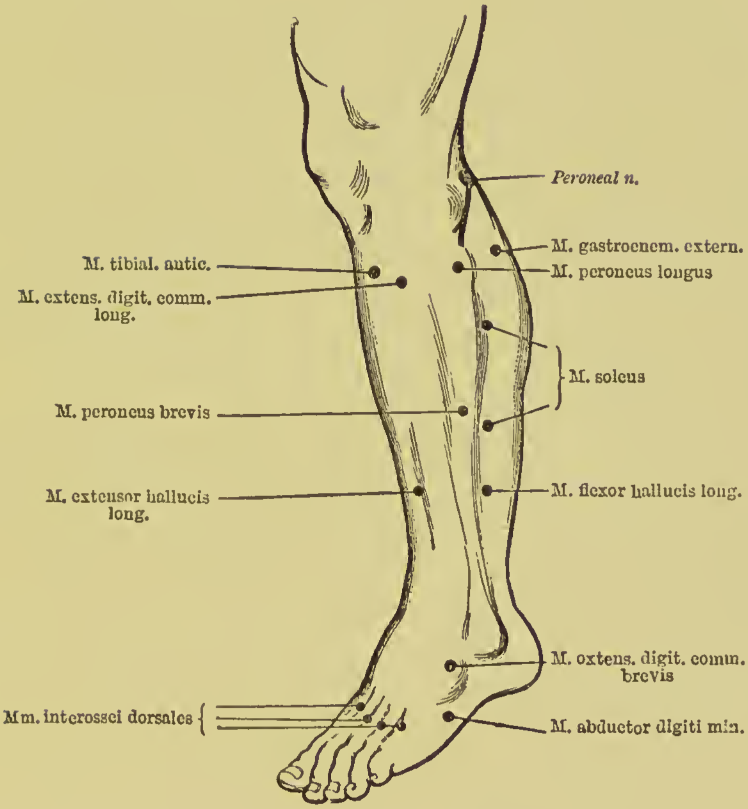
m. Extensor hallucis longus.

PLATE V


M. gluteus maximus
Sciatic n.
M. adductor magnus
M. semitendinosus
M. biceps. fem. (cap. long.)
M. semimembranosus
M. biceps fem. (cap. brev.)
Peroneal n.
Tibial n.
M. gastrocnem. (cap. extern.)
M. gastrocnem. (cap. int.)
M. soleus
M. soleus
M. flexor digitor. comm. longus
M. flexor hallucis longus
Tibial n.

m. Peroneus longus (Plate VI.).
m. Peroneus brevis.
m. Extensor digitorum brevis.
n. Internal popliteal.
(Motor point in principal transverse fold of popliteal space).
n. Posterior tibial.
m. Gastrocnemius.
m. Soleus.
m. Flexor hallucis longus.
m. Flexor digitorum longus.
(Motor point near inner border of tibia about middle of leg).
m. Abductor min. digiti.
m m. Interossei dorsales.

PLATE VI.


Peroneal n.
M. gastrocnem. extern.
M. tibial. antic.
M. peroneus longus
M. extens. digit. comm. long.
M. soleus
M. peroneus brevis
M. extensor hallucis long.
M. flexor hallucis long.
M. extens. digit. comm. brevis
Mm. interossei dorsales
M. abductor digiti min.

CHAPTER IX.

Electro-Cutaneous Sensibility.

Supplementary to the examination of the motor nerves is the investigation of cutaneous sensibility. It must not be expected that in electrical exploration will be found a means of determining the absolute sensibility of the skin as a sense organ. This must be ascertained by the application of appropriate stimuli.* Electro-diagnosis only offers itself as a means of estimating one kind of sensibility, viz., the sensibility of the skin to an electric current.† This form of sensibility may be increased, diminished, or abolished, and seems to correspond most closely to the sensibility to painful impressions.

The normal electro-cutaneous sensibility varies in different parts of the body. Duchenne well knew that a comparatively strong current is required to excite the sensibility of the foot or hand. This in his experiments was no doubt partly due to the greater resistance of the skin in those situations, a point which at the time in question was not fully realised. Still if the first minimal sensation and the first feeling of pain be tested by the application of a measured current it will be found that electro-cutaneous sensibility is by no means equally distributed over the body. Thus the anterior part of the body is more sensitive than the posterior. Anteriorly, sensibility is greatest about the face and neck, then come the arms and trunk and the lower extremity as far as the knee. The foot and leg come last, the heel requiring a current twice as great as any other part. Certain parts, such as the scrotum, the mamma, and wrist have a high degree of sensibility strictly limited to their own area.*

---

* For example, touch by the point of a pencil or by a shred of cotton-wool drawn lightly over the part ; heat by a cautery moved at a definite distance from the skin ; cold by a freezing mixture in a test-tube (this as well as the time honoured expedient of a hot and cold spoon becomes complicated with touch and pressure) ; pain by the "electric finger," &c.

† Erb.

The procedure to be followed in examining electro-cutaneous sensibility is much the same as that pursued in the examination of the motor nerve. It is obvious, that if an exact estimate is to be made, a current must be employed which is capable of exact measurement. This is not yet easy to do with the current of the induction coil. Therefore a continuous current, for the measurement of which every medical man who handles electricity now possesses an instrument, is best. Electro-cutaneous sensibility may be examined in several ways. The following is one method. Having applied the indifferent electrode (24 sq. in.) in the usual way, take a testing electrode, well padded but flat and about three-quarters of an inch in diameter, attach it to the negative pole, then with the circuit kept broken by means of the finger on the interrupter, throw a few cells into action and keep making closures as in testing a motor nerve. But in this case, instead of watching for the first contraction, ascertain the point at which the first closure becomes perceptible to the patient† and note the galvanometer. Another method is to proceed as follows:—Well wet and lightly dry the part to be examined, then with the indifferent electrode as before let the testing electrode rest on the part for one minute before, the current is allowed to pass. Commencing then with the handle of the cell collector at zero, gradually increase the electromotive force until the first sensation is felt by the patient. Read the galvanometer, then continue to throw in more cells, and note at what strength the first *painful* sensation appears. Do the same with the positive pole. It will thus be found that the negative pole (as in the case of the motor nerves) is the stronger stimulus. By a method similar to this it has been found (Bordier) that the electro-cutaneous sensibility of various parts of the body comes out as follows:—

---

* Bordier.

† The patient's eyes being closed and the examiner being very careful to keep the electrode steady in marking closures and openings.

Temple	0·3 m.a.
Shoulder	1·5 ,,
Forearm (anterior) . . .	1·0 ,,
,, (posterior) . . .	1·1 ,,
Hypothenar eminence . .	1·8 ,,
Foot (dorsum)	2·0 ,,
,, (heel)	5·0 ,,

Here, as in all electrical exploration, care must be taken to secure the same experimental conditions. As already said, the skin over the part examined must be carefully wetted and lightly dried, the electrode must be applied with the same amount of pressure and for the same length of time before the current is turned on, and on the appearance of the minimal sensation (or in the second method this and the feeling of pain respectively), the galvanometer must be immediately read.

In cases where the patient's answers are "suspect," it will be found a useful expedient to test with "the electric finger" and the Faradic current. The testing electrode instead of being applied to the patient is held by the operator, and the latter completes the circuit by applying a finger of his other hand to the patient's body. A means is thus secured of gauging the strength of the current, as well as the accuracy of the patient's replies.

Diagrams are added (pp. 59 to 63) showing roughly the topography of the sensory nerves. But it must not be supposed that each region of distribution is separated from its neighbour by any such clear line of demarcation. As a matter of fact careful testing will usually disclose an "overlap," *i.e.*, a limited region which derives its supply from two contiguous nerves. If, therefore the function of one of them be in abeyance, there is a region of only diminished sensibility on the confines of its territory.

Body Resistance in Diagnosis.

It is well known that in certain cases of hysteria the electrical resistance of the body is often very high and in others, *e.g.*, in exophthalmic goitre, it is often very low. It has therefore been considered that such facts can be turned to account in diagnosis; that they, for example, might be of service in the discovery of malingering, or useful to strengthen an opinion otherwise arrived at. But it would appear that body resistance is under the influence of causes so very variable and so little known that such opinions will not stand the test of experiment. Circulation and the functional activity of the nervous system, the general state of nutrition, the greater or less quantity of sodium chloride, are amongst the recognised disturbing causes, and there are doubtless others less well known. Spehl and Sano affirm as the result of 264 experiments on invalids and others:—(1). That the most diverse conditions both in health and in disease may present the same body resistance. (2). That whilst certain persons display an absolutely uniform electrical resistance to continuous currents, others both amongst the healthy and the sick show variations even during the course of the same day. (3). That the same disease presents different resistances in different persons and variable resistances in the same person.

Electro-muscular sensibility is discussed in Chapter XI.

PLATE VII.

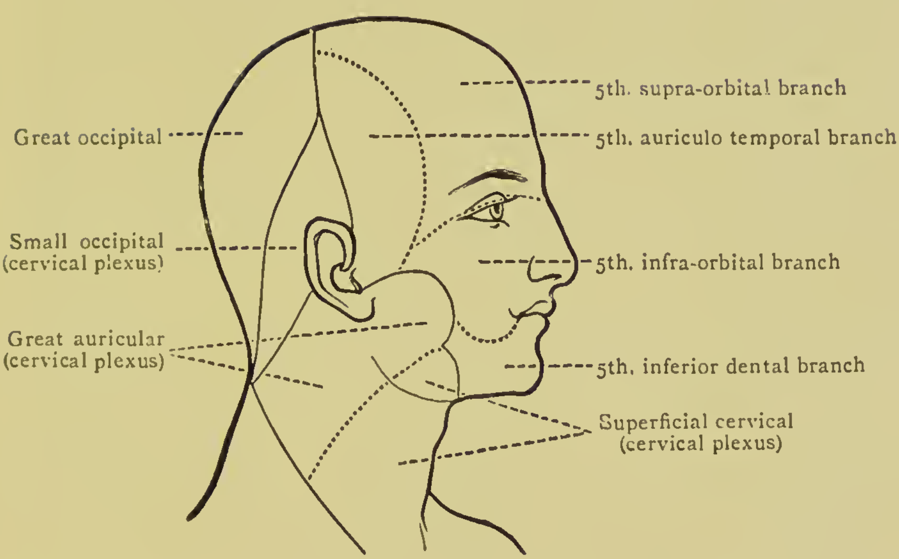

Great occipital
Small occipital (cervical plexus)
Great auricular (cervical plexus)
5th. supra-orbital branch
5th. auriculo temporal branch
5th. infra-orbital branch
5th. inferior dental branch
Superficial cervical (cervical plexus)

PLATE VIII

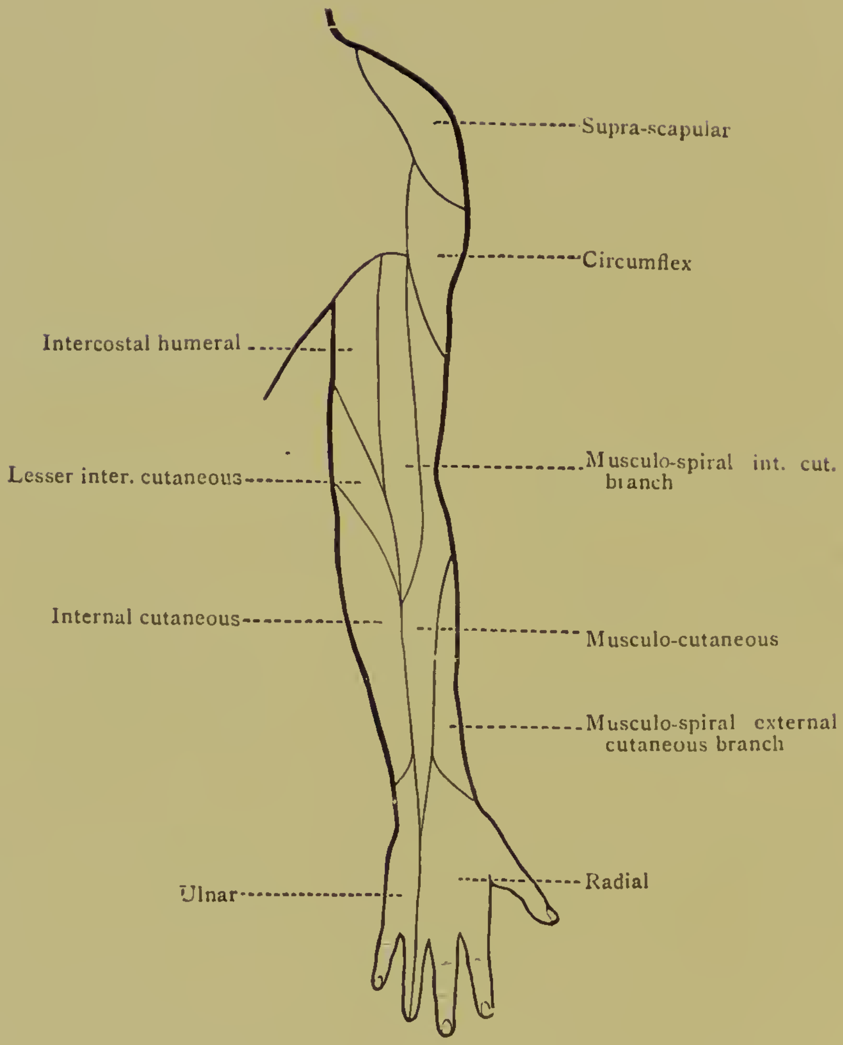

Supra-scapular
Circumflex
Intercostal humeral
Lesser inter. cutaneous
Musculo-spiral int. cut. branch
Internal cutaneous
Musculo-cutaneous
Musculo-spiral external cutaneous branch
Radial
Ulnar

PLATE IX.

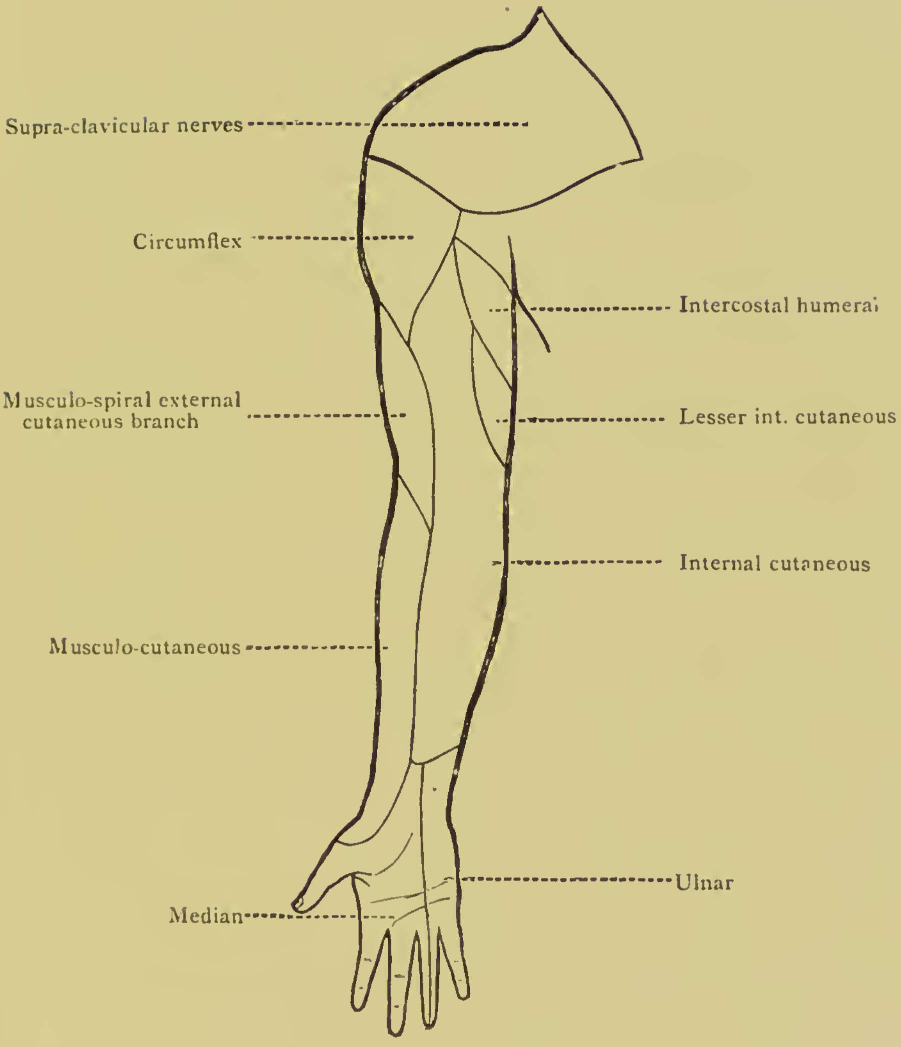

Supra-clavicular nerves
Circumflex
Intercostal humeral
Musculo-spiral external cutaneous branch
Lesser int. cutaneous
Internal cutaneous
Musculo-cutaneous
Ulnar
Median

PLATE X.

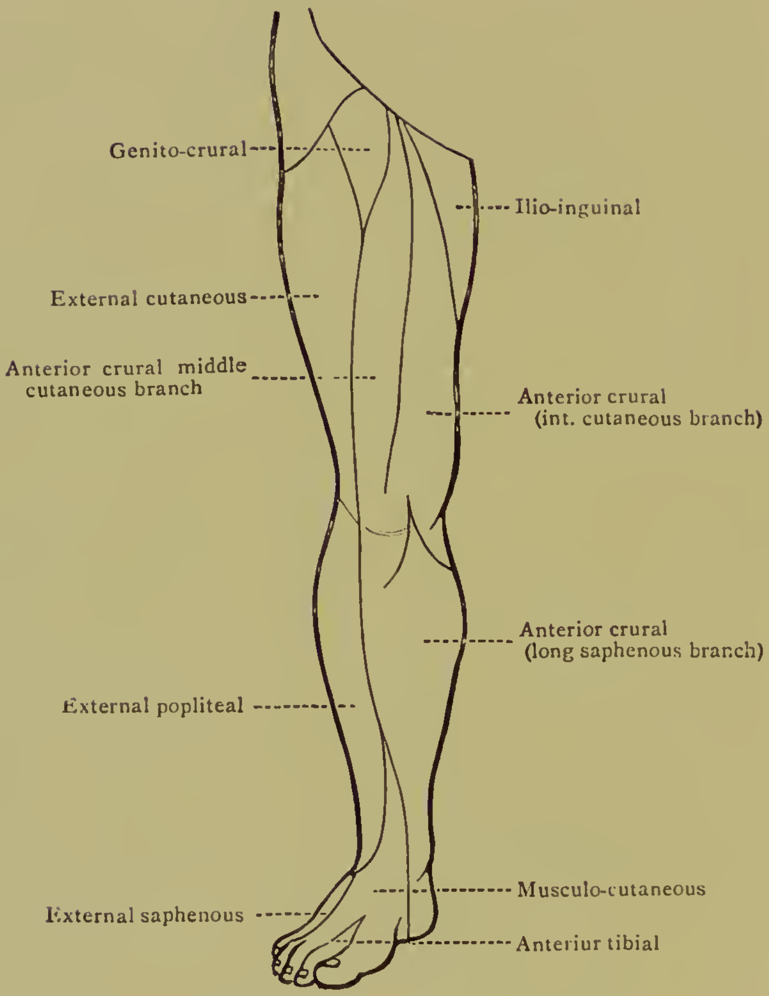
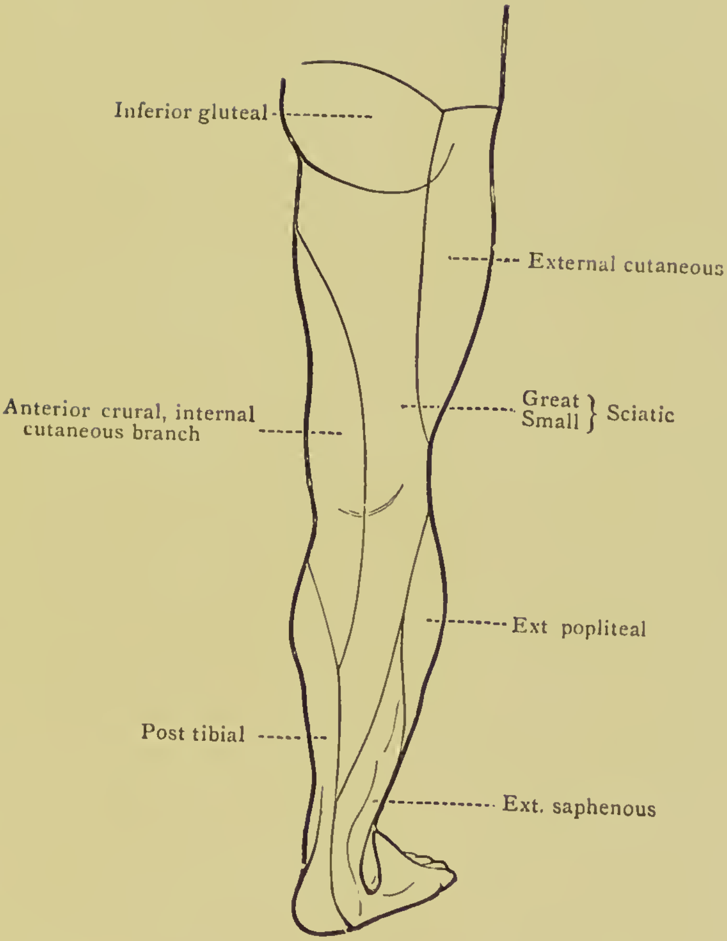

Genito-crural
Ilio-inguinal
External cutaneous
Anterior crural middle cutaneous branch
Anterior crural (int. cutaneous branch)
Anterior crural (long saphenous branch)
External popliteal
Musculo-cutaneous
External saphenous
Anteriur tibial

PLATE XI.


Inferior gluteal
External cutaneous
Great
Small
Sciatic
Anterior crural, internal cutaneous branch
Ext popliteal
Post tibial
Ext. saphenous

CHAPTER X.

THE ACTION OF MUSCLES AND THE CONSEQUENCES OF THEIR PARALYSIS.

ANY attempt to get exact ideas of voluntary movements by studying the isolated action of muscles, must at best result in comparatively crude conceptions. In every movement that a limb performs, its entire musculature perhaps co-operates, either in synergic or in antergic* action. Further, the mechanical effects of a muscle's contraction must vary with the varying positions of the articulations over which that muscle acts. For example, with the knee-joint bent, the gastrocnemius can no longer produce "plantar flexion" of the foot. Its origin and insertion have been too closely approximated. It has too much "slack" to take in. This is "active insufficiency." Under such conditions it is the soleus alone that pulls upon the tendo achillis to produce the movement in question. Again, with the knee extended the gastrocnemius is too short to permit complete "dorsal flexion" of the foot. This muscle stretched across the articulation, like a rigid cord, limits the action of the muscles that flex the tarsus upon the leg. This is "passive insufficiency."†

An analysis of the five following positions may be of use in roughly recalling the mechanics of muscular action.*

---

* The simultaneous contraction of opponents. "This is synchronous but not synergic." (Gowers).

† Landois and Stirling.

Figure 3.

Latissimus dorsi is in action attempting to force the arm downwards and backwards against resistance from above, at the same time tending to rotate it inwards. The *teres major* assists in both actions.

(When both *latissimi* act together they extend the trunk. The latter is inclined to one side if only one act. The latissimi are the great propelling muscles in swimming).

In paralysis of latissimus dorsi forcible backward depression of the raised arm is lost, and the shoulder cannot be put back without being also raised (by the trapezius).†

(The *trapezius* has three parts ; an " upper " or respiratory portion, a "middle" and a "lower." The two latter bring the scapula to the spine and put the shoulder back. The scapula and shoulder are elevated chiefly by the middle portion.

The *Deltoid* "abducts the humerus but does not raise the arm higher than to a right angle with the body ; the anterior and posterior fibres also move the arm forwards and backwards respectively.‡)

The *serratus magnus* is in strong contraction, fixing the scapula to the chest and assisting the trapezius (by rotating the scapula) to raise the shoulder above a right angle. (" If the scapula be fixed by the rhomboids, the serratus can act on the ribs and aid forced inspiration.")§

Paralysis of the serratus magnus does not lead to much

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* In actual testing the resistance would usually be supplied by the hands of the examiner.

† " Diseases of the Nervous System." Gowers.

‡ *Ibid.* § *Ibid.*

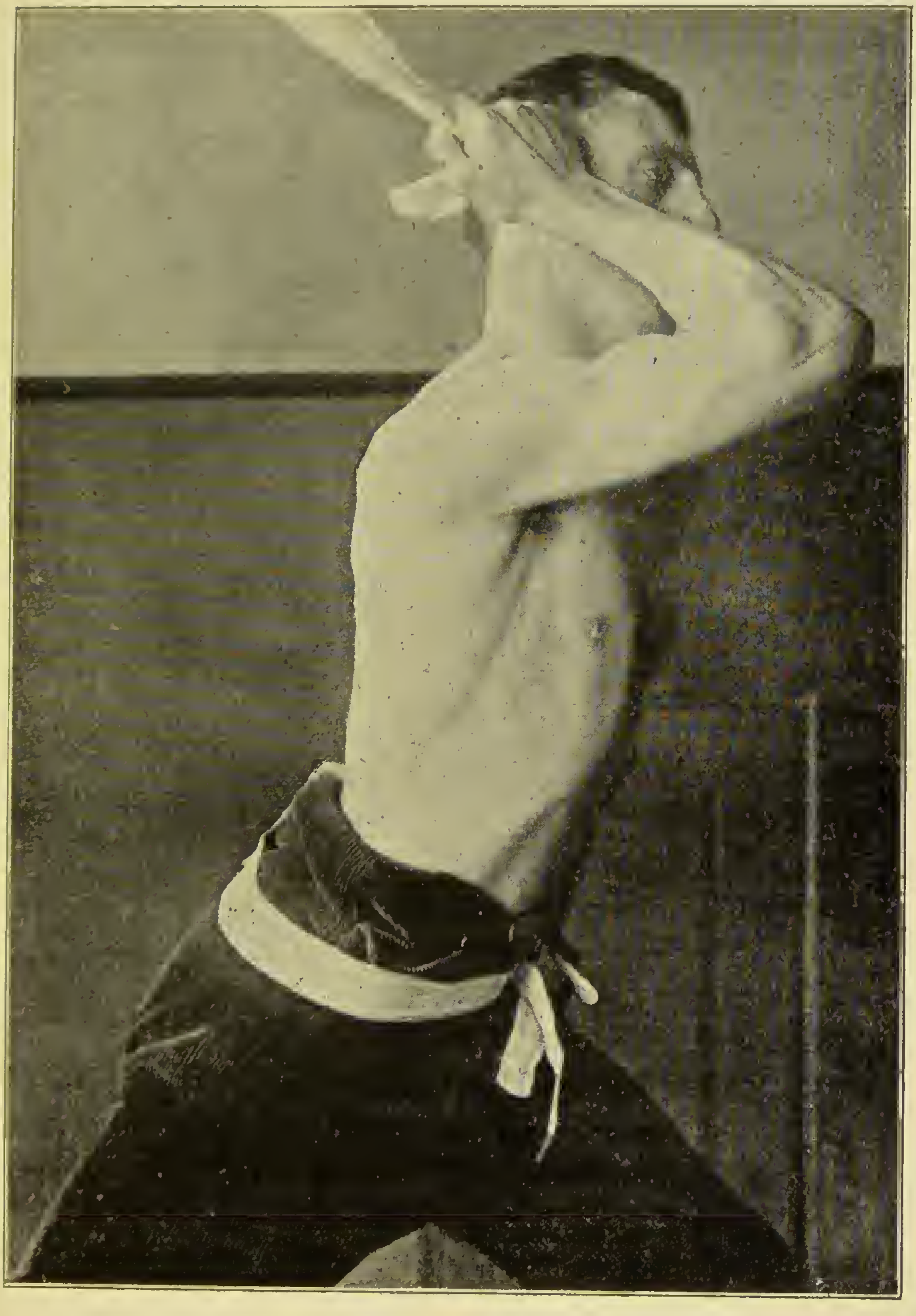

FIG. 3.

deformity when the scapula is at rest, but when the arm is moved forward by the anterior fibres of the deltoid, the scapula no longer held in position against the chest by the serratus magnus* is rotated on its vertical axis by the action of the anterior part of the deltoid on the humerus, and of the middle part on the scapula; the posterior edge, therefore, recedes from the thorax. The characteristic deformity thus produced is shown in fig. 4.

The *biceps* assisted by *supinator longus* is flexing the elbow, and fixing it by acting (in antagonism) with the triceps.

(" The *biceps* first supinates the forearm if it is pronated, then flexes the elbow."†

In paralysis the flexion can still be effected by other muscles).

The *supinator longus* "places the forearm midway between pronation and supination and then flexes the elbow."

(The *triceps* acting throughout its three parts extends the elbow.

" In paralysis of the triceps extension against gravity is impossible. A man with paralysis of the triceps cannot raise his hat in the accustomed manner "‡).

The *carpal extensors* by fixing the wrist are acting " in co-operative antagonism " to enable the flexing muscles to grasp the cord.

(" The *extensor carpi radialis brevior* is a direct extensor, the *extensor carpi radialis longior* and *extensor carpi ulnaris* move the hand laterally as well ")§.

(Paralysis of the extensors impairs flexion of the fingers by the too great approximation of the origin and insertion of the flexor muscles. They have too much "slack" to take in).

---

* *Ibid.* † *Ibid.* ‡ *Ibid.* § *Ibid.*

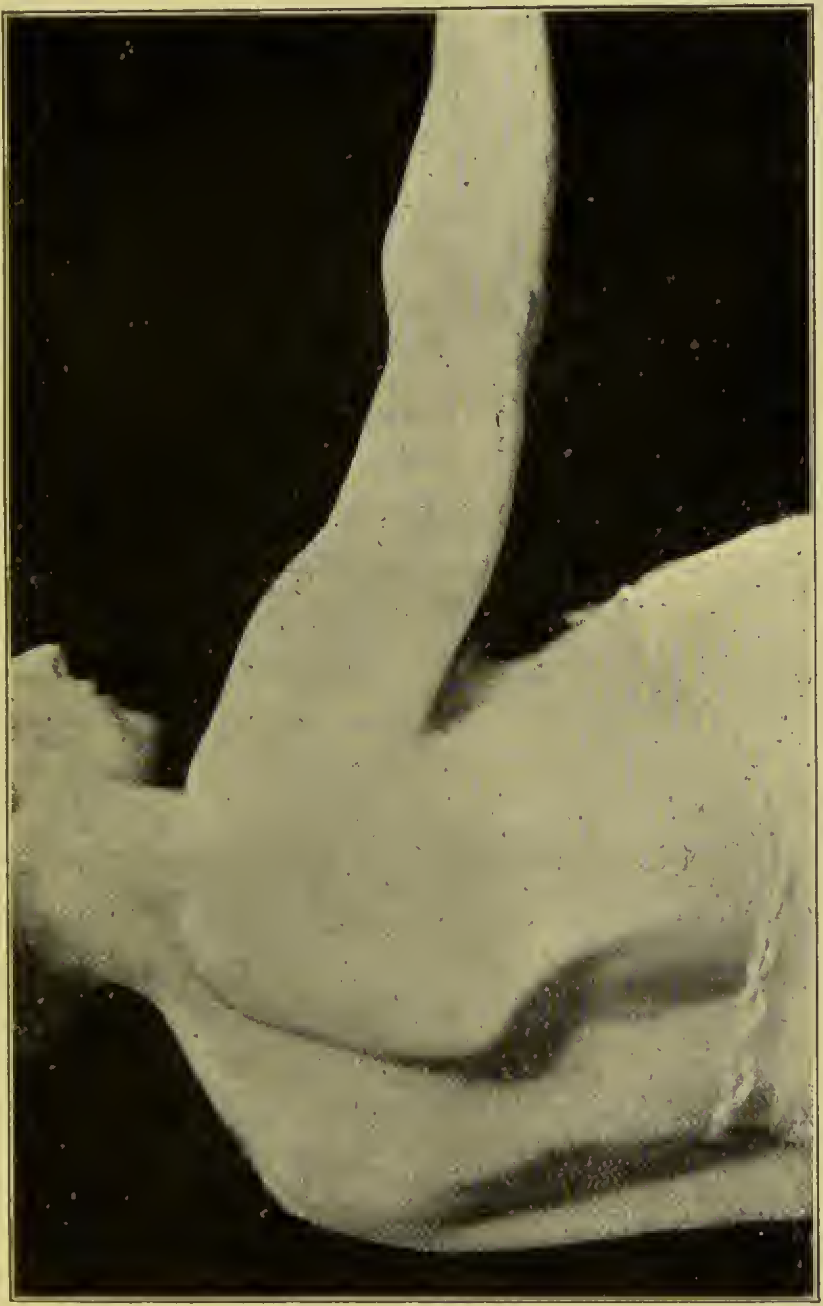

FIG. 4.

The *long flexors of the fingers* (flexor sublimis and flexor profundus) act chiefly on the second and third phalanges.

The *long extensors of the fingers* extend only the proximal phalanx.

The *interossei* fill up the gaps in this flexor-extensor action, that is to say they flex the first phalanx and extend the last two phalanges. They also adduct and abduct the fingers.

The *lumbricales* "aid the flexor-extensor action of the interossei, but do not move the fingers laterally."*

In paralysis of the interossei the long extensors, no longer opposed, extend the first phalanx permanently, and the long flexors, also unopposed, flex the second and third phalanges, producing the claw like attitude known as *main en griffe*.

Figure 5.

The *pectoralis major* is in action on both sides. Its upper portion bringing the shoulder forward and tending to raise it, the lower portion attempting to draw each arm to the side and across the chest, and at the same time tending to depress the shoulder. By pressing the hands together both arms are brought into action against resistance.

The *sterno-mastoid* inclines the head towards and rotates the head from the side on which the muscle contracts. Both muscles acting together bring the head forward to the vertical position. If they depress the head upon the chest they probably do so by acting upon the neck, the head being fixed.

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* *Ibid.*

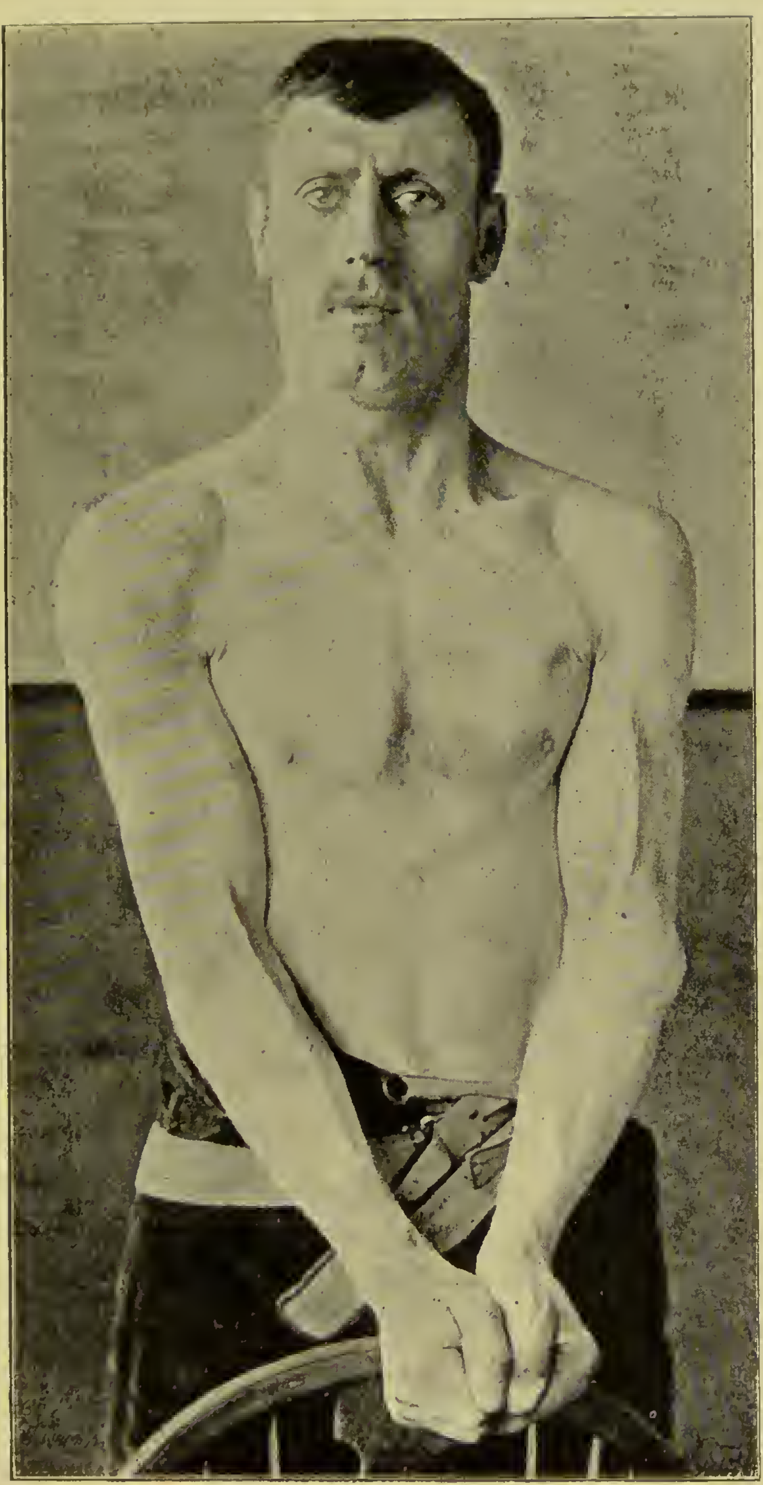

FIG. 5.

Paralysis of one muscle has no influence on the position of the head, and but little on its movements. Other muscles supplement the loss. There is no such thing as "paralytic torticollis" (Gowers).

Figure 6.

The arm is raised from the side of the body against the resistance of the weight of the chair by the action of the whole mass of the *deltoid*. The shoulder is elevated by:—

Trapezius and *serratus magnus* (rotating the scapula).

The *biceps* and *supinator longus* are flexing the elbow.

The *adductors of the hand* and *carpal extensors* are taking their share in adduction of the hand, and fixing the wrist in co-operative antagonism with the flexors and interossei by whose action the chair is grasped.

The right side of the body being in action, the head is turned to the same side by the left sterno-mastoid; and it is because the sterno-mastoid associates itself in action with the opposite side of the body that it is called a "contra-lateral muscle."

Figure 7.

The *quadriceps* is extending the knee. (The *rectus* may aid the *psoas* and *iliacus* in flexing the hip, but chiefly when the knee is bent. The *vasti* act only on the knee).

The *biceps, semitendinosus* and *semimembranosus,* are assisting in fixing the knee. They are flexors of the knee and extensors of the hip in ordinary walking. When a special effort is made the *gluteus maximus* is called into action for extension. The leg is rotated outwards by the biceps, inwards by the semitendinosus.

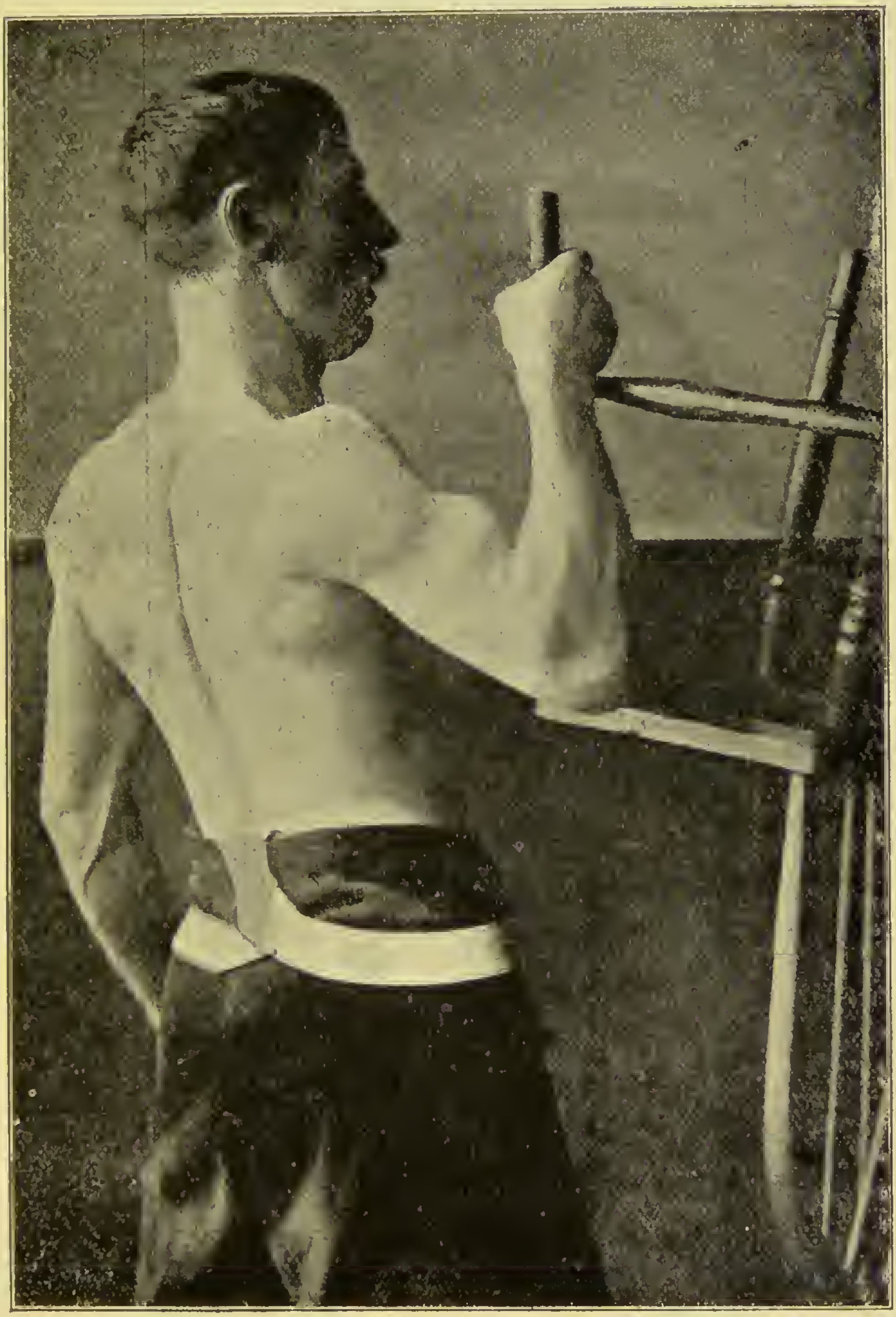

FIG. 6.

"In paralysis of the extensors of the knee, standing is

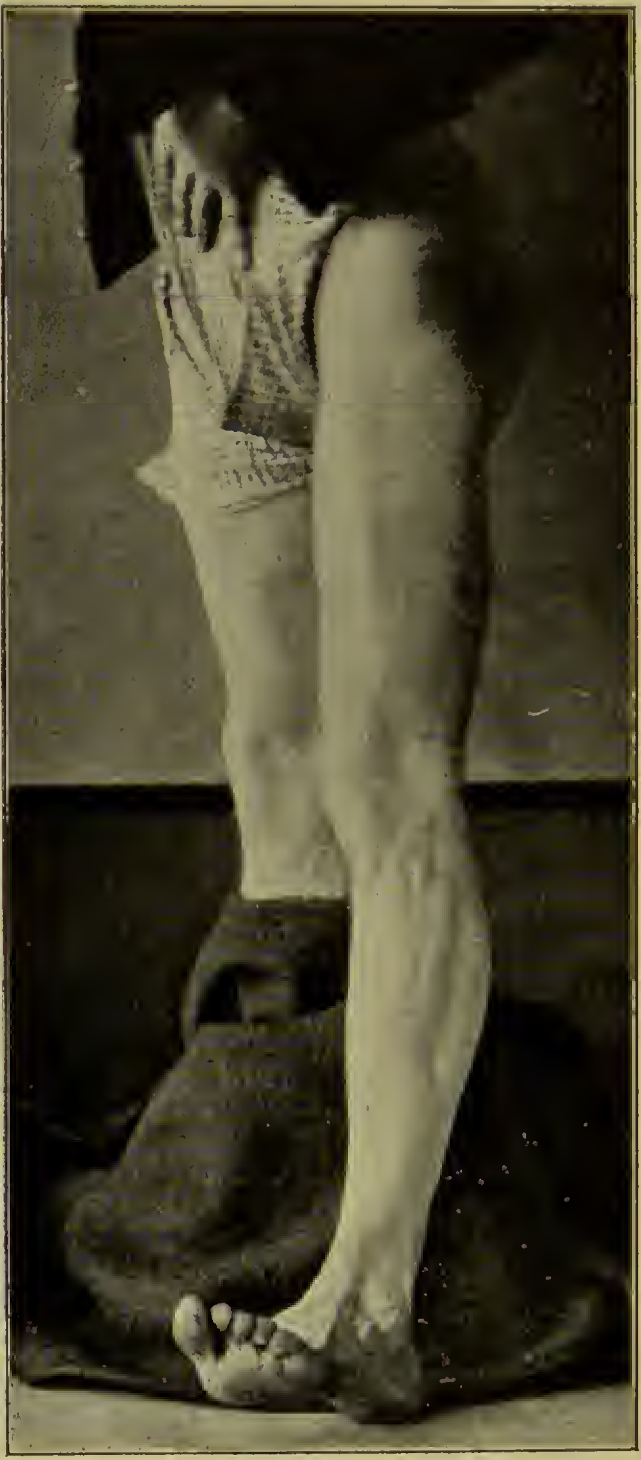

FIG. 7.

still possible if the knee is extended. Rising from the kneeling position is impossible."*

"In paralysis of the knee flexors the loss of power of flexion interferes with walking."†

The *tibialis anticus* and *extensor longus digitorum* flex the ankle. The former of these muscles is also an adductor, and the latter an abductor; acting together they flex the foot.

Paralysis of either "weakens flexion" and the corresponding lateral movement is lost. Secondary contracture of the extensors follows paralysis of the flexors, and *talipes equinus* is the consequence.

The *extensors of the ankle* are the calf muscles‡ (which also adduct), and peroneus longus (which also abducts).

Adduction and abduction, independently of flexion and extension, are carried out by the tibialis posticus and peroneus brevis respectively. Therefore:—

Paralysis of tibialis posticus produces *talipes valgus*, and paralysis of peroneus brevis produces *talipes varus*.

The *gastrocnemius* extends the hinder part of the foot and draws down the outer side. The *soleus* does the same, the only difference being that the latter "having no attachment to the *femur* can extend the ankle when the knee is flexed as well as when it is extended."§

The *intcrossei* act in a similar way to the interossei of the hand.

Of other muscles whose action is not brought out in the photographs the following points have to be remembered:—

Gluteus maximus extends the hip and rotates the thigh outwards. Is employed in going upstairs or rising from the sitting posture. Not much used in standing nor in ordinary walking.

---

* *Ibid.* † *Ibid.* ‡ *Ibid.* § *Ibid.*

Gluteus medius abducts. Its anterior third also moves the thigh forwards and rotates it inwards ; the posterior third effects the opposite movements.

In paralysis these movements are lost, and the unopposed action of the external rotators produces a permanent rotation outwards.

The *pyriformis, gemelli, obturator internus* and *quadratus*, all rotate the thigh outwards.

In paralysis the unopposed action of the internal rotators turns the leg and foot inwards.

The *adductor longus* (and the pectineus) produces an oblique movement forwards and inwards, *i.e.*, combined flexion and adduction as in crossing the legs. It also rotates outwards.

The *adductor magnus* causes a similar adduction and rotation. But it is stated* "that while its upper fibres rotate outwards its lower fibres rotate inwards, and are employed in keeping the foot straight during adduction in riding." It is easy to see that this would be the case were the leg extended at the knee ; but the latter can scarcely be called the "normal" riding position,—unless perhaps in the country of Duchenne.

The *innervation* of the muscles is given regionally with the motor points.

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* *Ibid.*

CHAPTER XI.

DEFINITIONS, EXPLANATIONS AND MEMORANDA.

PARALYSIS. Loss of power in a muscle may be partial or complete. Paralysis, which is medically applied to every degree of loss of power, is therefore distinguished as partial or complete.*

The strength of a muscle may usually be roughly tested by the examiner himself opposing its physiological action. With practice any considerable loss of power can thus be gauged with some accuracy, and especially so if a corresponding sound muscle be available for comparison. In doing this it is necessary to remember that the muscular force of the right side is usually greater than that of the left.†

Flexion of the fingers, *i.e.*, the "grasp" or "squeeze" may be measured by the dynamometer. The mean of several readings ought to be taken. The "pull" may also be measured.

Muscular sensibility.—The muscles are supplied by sensory nerves, and pain is caused on the application of too strong a stimulus, as in the case of other sensory nerves. When a muscle is made to contract by an electric current the contraction is felt. With a strong stimulus a feel-

---

* The term "paresis" is sometimes used instead of slight paralysis. Kinesis and akinesis are sometimes used to denote movement and loss of movement respectively.

† In the proportion of five to four (Gowers).

ing of deep tension—a "drawn" feeling—is produced amounting to pain. The complication of electro-cutaneous sensibility with this feeling can to some extent be eliminated by anæsthetising the skin; or by making the muscle contract through its nerve trunk, instead of by a direct application to the muscle. The pain is presumably caused by compression on the nerve endings by the muscular contraction. Can electro-muscular sensibility be excited *without* a contraction, *i.e.*, by direct stimulation of the sensory nerves? A case combining the absence of muscular contractility on electrical stimulation with complete anæsthesia of the skin would afford an opportunity of demonstrating this point. But it is quite certain that electro-cutaneous and electro-muscular sensibility are distinct. In disease, one may be absent and the other present. The power of discriminating differences in weight is the recognised test of muscular sensibility. The knowledge which a person has, unaided by other senses, of his own posture whether active or passive, *i.e.*, either when his muscles are active in obedience to the will or only in the "slighter activity" of rest, is ascribed to the "muscular sense," a term which therefore has a more restricted meaning than muscular sensibility.*
The latter as above described includes ordinary muscular sensibility to stimuli, and pain when such stimuli are excessive.

Reflex action.—Defined as an action that is "effected through an afferent sensory nerve, an efferent motor nerve, and a system of nerve cells and fibres connecting the two in the spinal cord, *the reflex centre*. . . . . The reflex centre is between the roots of the sensory and motor nerves concerned and with them constitutes what may be called *a reflex arc*."* There are two chief forms of reflex action :—(1) cutaneous reflex ; (2) muscle reflex.

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* See Gower's "Diseases of Nervous System," p. 14.

(1). "Cutaneous reflex," "superficial reflex"—excited by stimulation in the form of stroking the skin adjacent to the muscle tested. The *plantar* reflex is produced by tickling the sole of the foot ; this excites slight movement of the toes (through 1st, 2nd, and 3rd sacral). The *gluteal* obtained by stimulating the skin of the gluteal region (4th and 5th lumbar nerve) ; *cremasteric* by stimulating skin of the upper and inner part of the thigh producing retraction of the testicle (1st and 2nd lumbar) ; *epigastric* by stimulating the skin of the chest over the fifth and sixth intercostal spaces, producing dimpling of epigastrium (4th, 5th, 6th and 7th dorsal nerves) ; *scapular* by interscapular stimulation of skin producing contraction of scapular muscles and those of the posterior fold of the axilla. Of these the plantar reflex is the least seldom absent in health. The *conjunctival* reflex and the *contraction of the iris* from slight and its dilatation from strong stimulation of neck, are also examples of superficial reflexes.

(2). "Muscle reflex"—"tendon reflex"—"deep reflex," excited by "a peculiar mode of stimulation of deeper nerves, probably those only of the muscles" (Gowers).

These "reflexes" are produced by the sudden increase of tension in muscles which are already in a state of slight tension. The patellar tendon reflex or knee-jerk is elicited by flexing the knee "so that the quadriceps is gently extended and the leg free to move," and then suddenly striking the tendon of the patella. The contraction of the quadriceps jerks the knee forward.* The normal knee-jerk depends upon the integrity of the cord at the level of the 2nd and 3rd lumbar nerves, and on the normal condition of the other constituents of the reflex arc.

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* *Ibid.*

The physiology of the knee-jerk has been much discussed. Gowers suggests that the passive tension excites in the muscle, by reflex influence, a state of irritability to local mechanical stimulation, such as a sudden tap. It is not stimulation of the nerves of the tendon that induces the contraction, "but the stimulus originates in the muscle." The tendon acts mechanically on the muscle. It is the string, so to speak, which pulls upon the muscle and excites it.

A phenomenon similar to the above, occurs at the ankle. If the calf muscles are made tense and the tendo Achillis or muscles on the front of the leg be tapped, a slight extension of the foot is produced. In certain pathological conditions, if the ankle is forcibly flexed by pressing the hand against the sole of the foot, a clonic series of contractions occur, known as "ankle clonus." It depends upon the condition of the cord at the level of the three first sacral nerves, and usually denotes structural changes in the lateral columns. " Front tap contraction," *i.e.*, the slight contraction of the gastrocnemius obtained by tapping the muscles on the front of the leg, does not always point to structural change in the cord. Percussion of the tendons of the flexors and extensors of the wrist, and of the tendon of the triceps, may produce muscular contractions. In health these are usually feeble or altogether absent.

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* To elicit the knee-jerk it is usual to let the patient sit on the edge of a table with the legs crossed at the knee, the leg to be examined being supported by the other. The knee of the supporting leg is at a right angle, the knee of the leg to be examined is flexed, but not quite to a right angle. The patient is then directed to interlock his fingers and pull in the direction of pulling his hands apart. Voluntary effort is thus taken off the leg. The patellar tendon is then struck with the ulnar border of the hand or with a percussion hammer. There is nothing better for this purpose than the rubber-edged ear-piece of the old stethoscope.

Diminution in the size of the muscles and alteration in their minute structure follow lesions of the lower segment of the motor path. The wasting of a muscle can to some extent be gauged by measurement. The minute changes of structure need not here be described. The changes in electrical reactions which correspond to these have been dealt with under electrical testing.

"Inco-ordination," "ataxy," has been defined as the inability to produce voluntary muscular movement in proper order or sequence. "It is not customary to apply the term to any derangement of movement except that which is irregular in time. . . . . It is necessary to distinguish co-ordination, and the *direction* of movement to a definite end. The motor process for the latter depends on true sensory guidance, chiefly on the senses of touch and sight. A sempstress with loss of sensation in the fingers cannot sew unless she constantly watches them" (Gowers).

Certain practical points in connection with spinal cord anatomy may here be called to mind.

It is customary to speak of "the thirty-one segments of the spinal cord"—a "segment" being that portion of the cord from which a pair of spinal nerves arise. The cord fills only the upper two-thirds of the spinal canal and extends from the foramen magnum to the upper point of the second lumbar spine. The relations of the spines to the vertebral "body" and to the origins of the "nerve-pairs" must be remembered. The "vertebra prominens," *i.e.*, the spine of the seventh cervical vertebra is opposite the first dorsal nerve roots. From the 3rd to 10th dorsal, the spines correspond to the second root below; the 11th spine corresponds to 1st and 2nd lumbar nerves; the 12th to the 3rd, 4th and 5th; the 1st lumbar to the 1st, 2nd and 3rd sacral nerves.* The upper part of 2nd lumbar is opposite the tip of the cord. When the spinous process is difficult to feel it is convenient to "follow the last rib to its junction with the vertebra" (Morton).

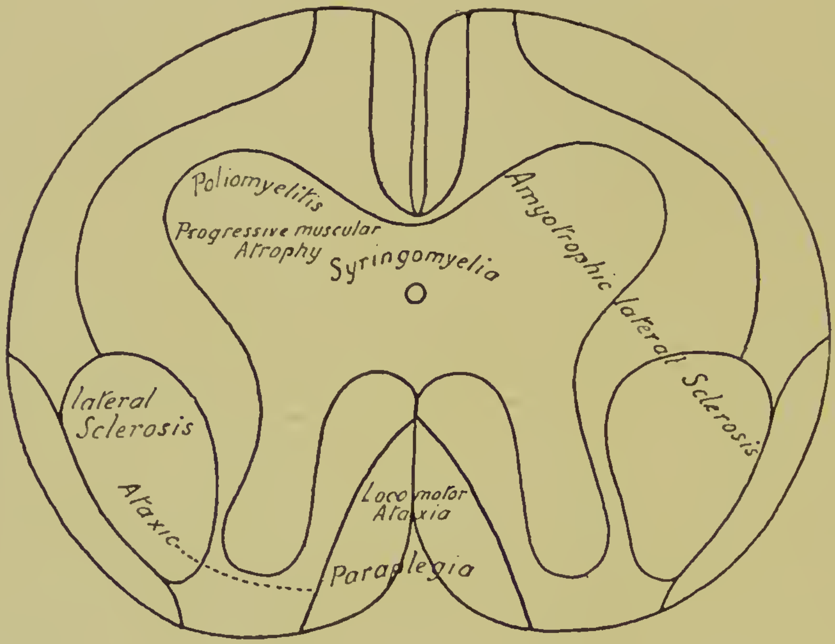

FIG. 8.

The lumbo-sacral enlargement begins in front of the tip of the spine of the 10th dorsal vertebra. The cervical enlargement is above the 7th cervical. Each segment comprises in itself a motor, a sensory, and a reflex mechanism, and has a corresponding representation in the periphery. It is largely through the latter that the spinal cord is influenced by electrical and other stimuli.

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* Gowers.

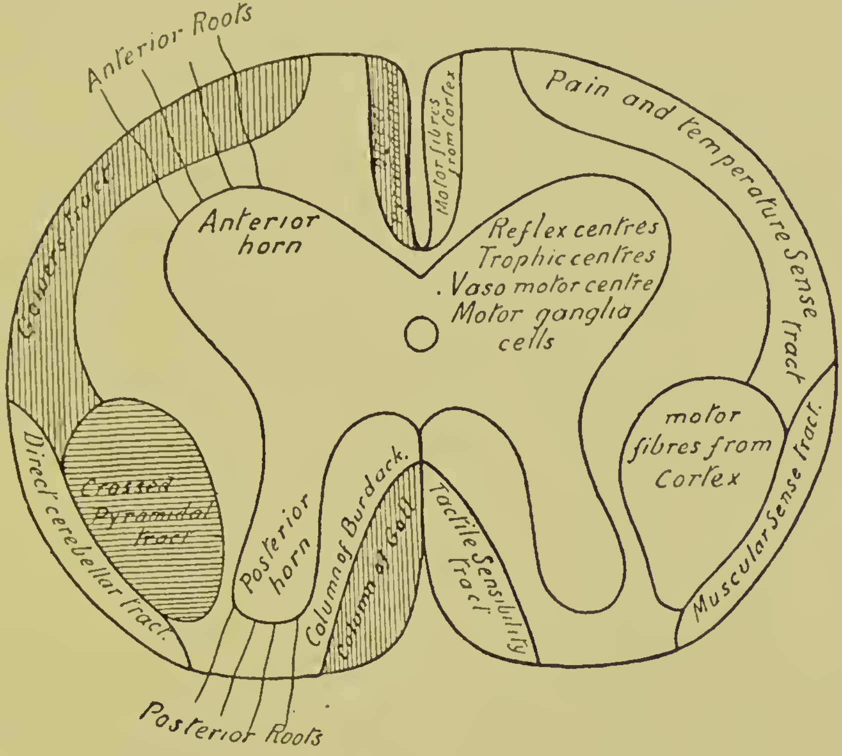

FIG. 9.

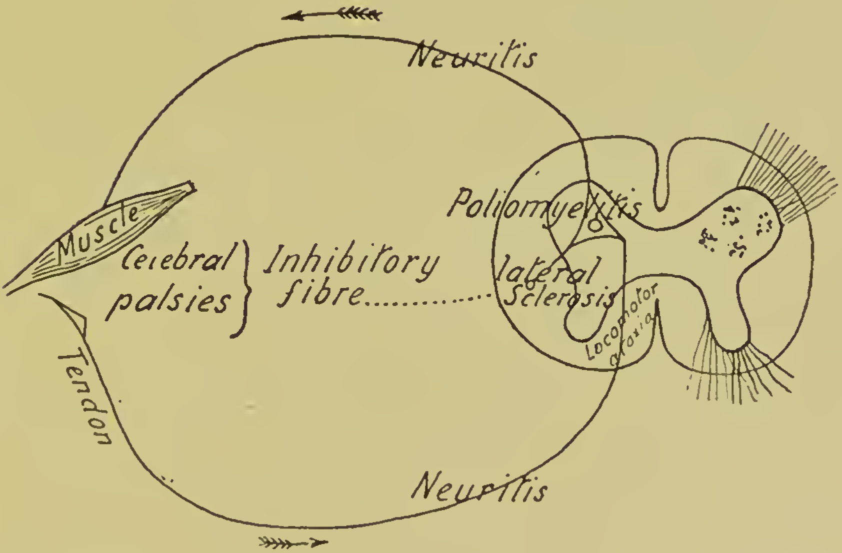

FIG. 10.

"*System diseases.*"—This term is applied to those pathological conditions which attack certain columns of the cord or certain parts of the grey matter, *i.e.*, certain "systems" of fibres or cells according to their functions.

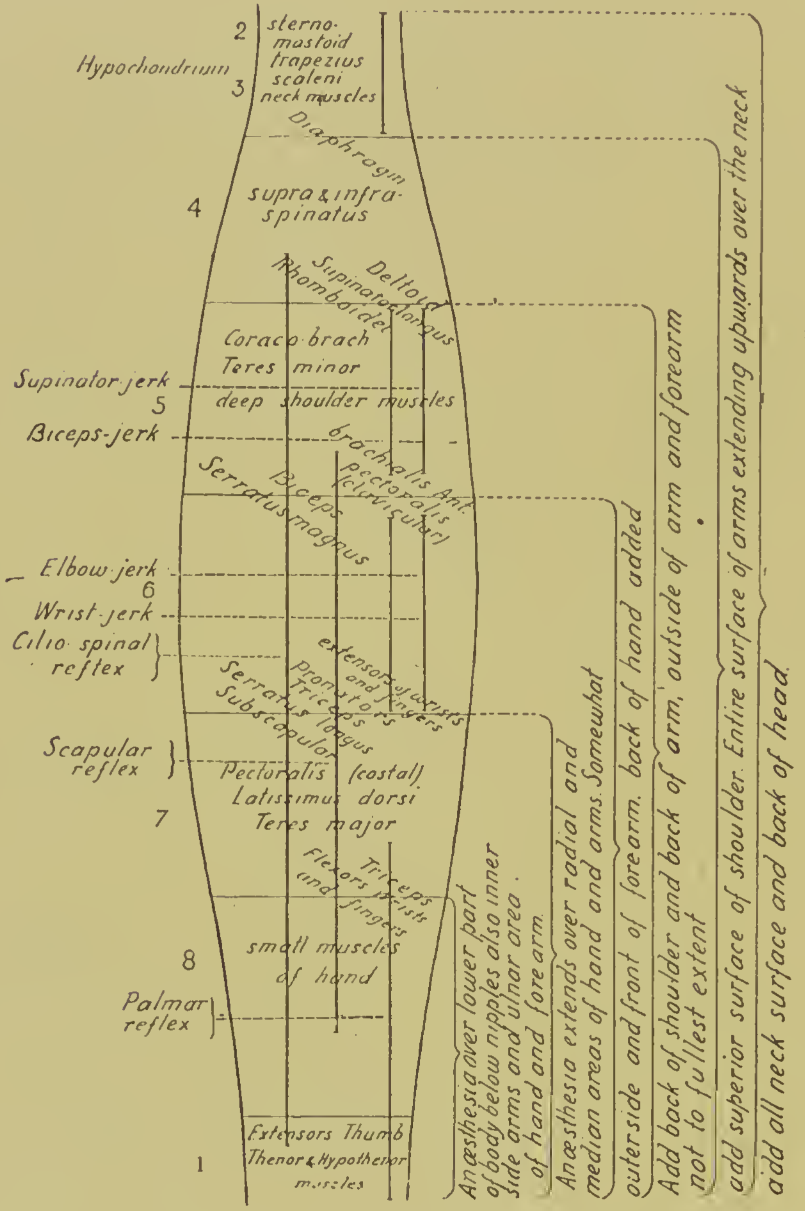
2
sterno-mastoid
trapezius
scaleni
neck muscles
Hypochondrium
3
Diaphragm
4
supra & infra-spinatus
Supinator longus
Deltoid
Rhomboideus
Coraco-brach
Teres minor
Supinator-jerk
5
deep shoulder muscles
Biceps-jerk
Brachialis Ant.
Pectoralis (clavicular)
Serratus magnus
Biceps
Elbow-jerk
6
Wrist-jerk
Cilio-spinal reflex
extensors of wrist and fingers
Pronators
Triceps longus
Serratus
Subscapular
Scapular reflex
Pectoralis (costal)
Latissimus dorsi
Teres major
7
Flexors of wrist and fingers
Triceps
small muscles of hand
8
Palmar reflex
Extensors Thumb
Thenor & Hypothenor muscles
1
Anæsthesia over lower part of body below nipples also inner side arms and ulnar area of hand and fore arm.
Anæsthesia extends over radial and median areas of hand and arms. Somewhat outer side and front of forearm. back of hand added
Add back of shoulder and back of arm, outside of arm and forearm not to fullest extent
add superior surface of shoulder. Entire surface of arms extending upwards over the neck
add all neck surface and back of head.

Fig. 11.

Poliomyelitis, lateral sclerosis, locomotor ataxy, progressive muscular atrophy, amyotrophic lateral sclerosis, ataxic paraplegia, are instances of these. Their localisation is roughly shown in fig. 8 (after Peterson). Certain points in the anatomy, physiology, and pathology of the cord are shown in figs. 9 and 10 (after Peterson).

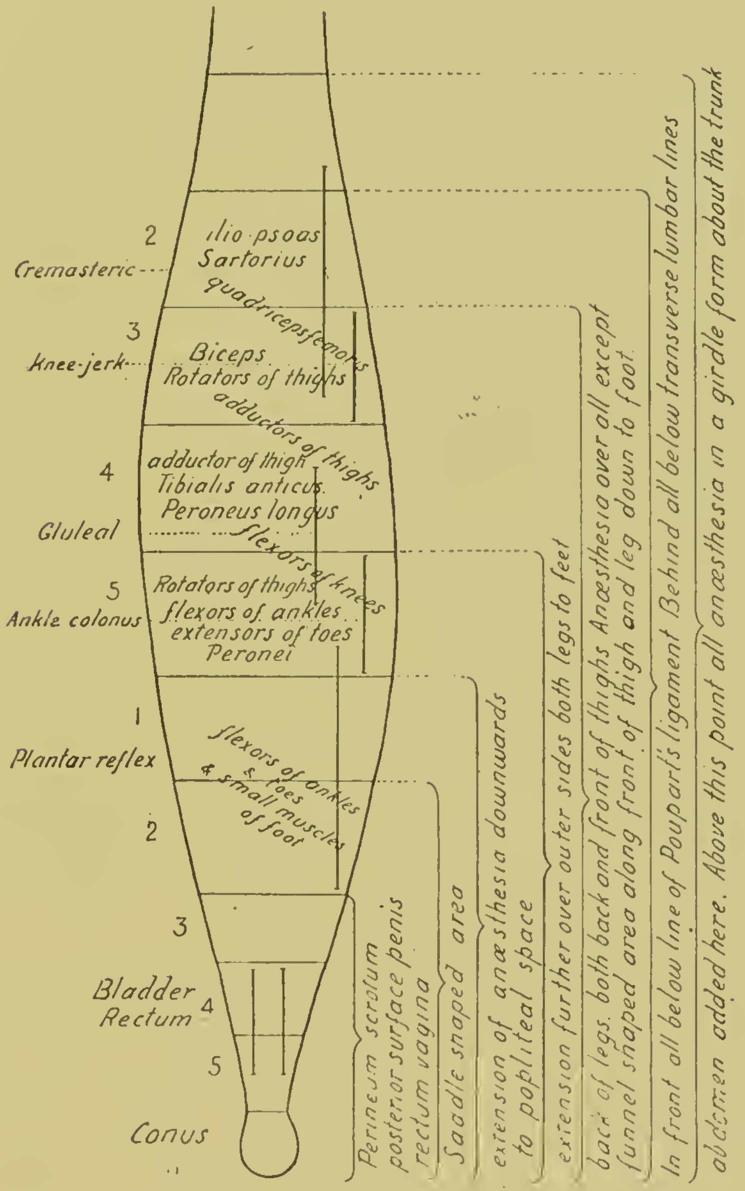
2
Cremasteric
3
Knee-jerk
4
Gluteal
5
Ankle colonus
1
Plantar reflex
2
3
Bladder
Rectum
4
5
Conus
ilio-psoas
Sartorius
quadriceps femoris
Biceps
Rotators of thighs
adductors of thighs
adductor of thigh
Tibialis anticus
Peroneus longus
flexors of knees
Rotators of thighs
flexors of ankles
extensors of toes
Peronei
flexors of ankles & toes & small muscles of foot
Perineum scrotum posterior surface penis rectum vagina
Saddle shaped area
extension of anæsthesia downwards to popliteal space
extension further over outer sides both legs to feet
back of legs. both back and front of thighs Anæsthesia over all except funnel shaped area along front of thigh and leg down to foot
In front all below line of Poupart's ligament Behind all below transverse lumbar lines
abdomen added here. Above this point all anæsthesia in a girdle form about the trunk

Fig. 12.

"Focal" diseases are lesions of limited extent in a particular segment of the cord. Spinal injury, inflammation, hæmorrhages, pressure, tumours, are instances of such, and the symptoms necessarily depend upon the level of the lesion. The distribution of the various reflex, motor, trophic and sensory functions of the cord is roughly shown in figs. 11 and 12 (after Peterson).

II.

THE TREATMENT OF MUSCULAR ATROPHIES.

TREATMENT OF MUSCULAR ATROPHIES.

CHAPTER I.

CLASSIFICATION.

THE field covered by the muscular atrophies is wide and difficult, and by no means fully explored. Yet in looking for a rational treatment it is necessary in the first instance to make a brief survey of some salient points in pathology and classification. In attempting this it may lead to clear ideas, first to define those atrophies which are known as "progressive muscular atrophies," and then to consider those which do not come under such a definition. The progressive muscular atrophies have been described as follows.*

"When a muscle, or a group of muscles, is diminished in volume, and when at the same time there is diminution of the muscular fibre under the microscope, it may be said that there is true muscular atrophy. When the volume of the muscle being preserved or augmented, histological examination shows a diminution of the muscular fibres, at the same time that there is hypertrophy of fatty and cellular tissue, this also is muscular atrophy (pseudo-hypertrophic); when in either case the number of muscles attacked by the atrophy progressively increases, when the affection shows no tendency to regression nor even to localisation, but when group by group the atrophy invades most of the striated muscles of the body, the condition is termed 'progressive muscular atrophy.'"

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* "Gaz. Med. de Paris."

Accepting this definition and taking advantage of the classification that accompanies it, muscular atrophies may for present purposes be thus tabulated :—

A. PROGRESSIVE MUSCULAR ATROPHIES.
  I. *Myelopathic.*
    1. Aran-Duchenne type.
    2. Charcot-Marie type.
  II. *Myopathic.*
    1. Charcot-Duchenne type, ("Pseudo-hypertrophic Paralysis").
    2. Landouzy-Dejerine type, ("Progressive Atrophic Myopathy").
    Other less important types are as follows:—
    3. Leyden-Möebius type.
    4. Erb type.
    5. Zimmerlin type.
    6. Mixed types.

B. MUSCULAR ATROPHIES, OTHER THAN THOSE KNOWN AS "PROGRESSIVE."
    1. Myelopathic atrophies in which the paralysis precedes the atrophy properly so-called, amyotrophic lateral sclerosis, diffuse myelitis, lateral sclerosis, compression of cord, tabes.
    2. Glosso-labio-laryngeal paralysis.
    3. Atrophies following lesions of nerves.
    4. Atrophic paralysis of Deltoid ("rheumatic").
    5. Lead palsy.
    6. Infantile paralysis.
    7. Peripheral neuritis.
    8. Atrophies following articular lesions.

Still following for the most part the classification referred to, the foregoing types are broadly differentiated as follows:—

A. I. *Myelopathic progressive muscular atrophy.* Primary, systematised, and progressive disease of anterior cornua; characterised clinically, (*a*) by qualitative modifications of electrical excitability (R.D.), (*b*) by tremors or fibrillary contractions. Two types, Aran-Duchenne and Charcot-Marie.

1. *Aran-Duchenne type.* A disease of adult life most frequent in males. Heredity and sometimes excessive muscular fatigue seem to be factors in its production. Pathologically it is a pigmentary degeneration or "sclerotic" atrophy beginning in the motor ganglion cells and invading the anterior roots. The muscle undergoes progressive diminution in volume but without fatty degeneration and its structure remains intact. The atrophy is due to the loss of the trophic influence of the motor ganglion cells in the cord.

Symptoms. This atrophy is in no sense a paralysis but a diminution in muscular power due to atrophy. The hand is first affected, often the right hand, but the disease soon becomes bilateral. The thenar muscles disappear. The unopposed muscles draw the metacarpal bone, outwards and backwards (*main de singe*). Then the interossei (flexors of 1st phalanx, extensors of the two last) disappear and the antagonistic muscles being unopposed, the result is extension of the first phalanx, flexion of the two last (*main en griffe*). Later, all the muscles of the hand are so much atrophied that the fleshless hand is like that of a skeleton (*main de squelette*).

It is noticeable that in the forearm the muscles on the anterior aspect are often attacked first. When the shoulder and trunk muscles are attacked, the Deltoid atrophies throughout, but the Trapezius only at the part supplied by the cervical plexus. Spinal curvature may be induced by these atrophies, and the muscles of the neck may become implicated. At this stage the intact condition of the lower extremities offers a striking contrast to the atrophy that prevails elsewhere. The lower extremities are not as a rule affected, and only at a late stage of the disease. Respiration and digestion are so far intact, but when the depressor muscles of the lower jaw and the muscles of deglutition and respiration become involved, the condition is of course very grave. There may be temporary arrest in the symptoms, but the course is slowly progressive. It may be a dozen years or so before the respiratory muscles are affected, and the duration of the disease is from 1 to 20 years. Death will often occur from some inflammatory complication of the respiratory passages when the muscles of inspiration are weakened and atrophied; but the disease itself may be the direct cause of death by asphyxia from atrophy of the diaphragm.

Complications. Glosso-labio-laryngeal paralysis, œdema, skin eruption, sweating. There may be sensations of cold, but no alteration in cutaneous sensibility.*

Two varieties.—(*a*) Where fatty tissue in the muscles may to some extent mask the atrophy.

(*b*) *Vulpian type,* (scapulo-humeral), begins with the muscles of the shoulder and arm, and is a long time before it spreads, and never attacks face muscles.

2. *Charcot-Marie,* begins at the feet—attacks in succession, legs, arms, hands and forearm. Extensors

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* "Gaz. Med. de Paris."

suffer more than flexors. Distinctly hereditary, and is accompanied by fibrillary contractions and vaso-motor troubles.

A. II. *Myopathic progressive muscular atrophy* (primitive progressive amyotrophies of Charcot). The feature common to all atrophies of this class is that there is no appreciable alteration of nerves or nerve centres. The myopathy seems to be "primary." They are all hereditary, or at least occur in members of the same family, and in early life. No R.D. No fibrillary contractions.

1. *Charcot-Duchenne type* (pseudo-hypertrophic paralysis). Disease of infancy or early years. Most frequent in boys. Often several members of a family attacked. There is an *apparent* muscular hypertrophy, at the same time that the muscular fibre becomes replaced by connective tissue and fat, but the muscular fibre is simply atrophied and seldom undergoes fatty degeneration; cord and nerve seem to be unaffected.

Symptoms. Muscular hypertrophy (apparent), generally begins in the muscles of the calf. Almost always progressive, but slow and without rise in temperature. Lasts many years (10 to 20).

2. *Landouzy-Dejerine type* ("progressive myopathic atrophy," "facio-scapulo-humeral"). May appear at any age. Most frequent in boys. As in the foregoing and other pure myopathies the essential lesion is atrophy of the muscular fibre, simple and primary. No lesion of nerve centres or nerves. Begins with face, attacking first the orbicularis oris and orbicularis palpebrarum. Patient cannot close eyes or mouth. After some years spreads to muscles of upper limbs and trunk. Trapezius, rhomboid, deltoid, biceps, brachialis, triceps, sup. long., and ext.

c. rad., are generally the only muscles affected for many years. The forearm retains its muscularity long after the arm muscles are atrophied. Progress slow, and gets steadily worse. Prognosis bad.

Less well marked types of the myopathies are as follows :—

3. *Leyden-Möebius type.* This follows the course of pseudo-hypertrophic atrophy without the hypertophic stage. Perhaps a transition form connecting the latter with Erb's type. It commences with the calves and follows a course irregularly ascending.

4. *Erb's type* ("Juvenile"). Begins generally in infancy or about puberty, usually in muscles of shoulder and arm; sometimes it commences in the muscles of the loins and lower extremity; muscles of the forearm remain a long time unaffected (except sup. long.), and muscles of hand scarcely ever attacked.

No R.D. No fibrillary contractions.

5. *Zimmerlin type.* Closely resembles the last.

6. *Mixed types.* Combinations and modifications of all the above.

Marie says that "in atrophy the volume of the muscle is nothing, the weakness is everything." Charcot says, "all these types belong to one species, primary progressive amyotrophy." The foregoing definition of "progressive muscular atrophies" distinguishes them from—

B. *Other muscular atrophies.*

1. *Myelopathies distinguishable from progressive muscular atrophies by the fact that the paralysis is the primary and leading feature and precedes the atrophy properly so-called.* (Amyotrophic lateral sclerosis, diffuse myelitis, lateral sclerosis, compression of the cord, locomotor ataxy).

2. *Glosso-labio-laryngeal paralysis* is a *local* progressive atrophy which commencing at the tongue attacks the lips, soft palate, and larynx. Here again the atrophy is *secondary*.

3. *Atrophies following lesions of nerves.* The history of an injury to the nerve, the localisation of the atrophy to the distribution of that nerve, the pain and anæsthesia all serve to distinguish such an atrophy from one which would come under the term "progressive."

4. *Atrophic paralysis of deltoid* ("rheumatic") is accompanied by pain.

5. *Lead palsy.* Characterised primarily by paralysis, and it falls chiefly upon the extensors. Atrophy does not supervene until late. Supinator longus usually escapes.

6. *Infantile paralysis.* Its course is the reverse of a progressive muscular atrophy. Paralysis complete from the first, but a tendency to spontaneous recovery.

7. *Multiple peripheral neuritis.* The early paralysis, its greater incidence on the extensors, the sensory phenomena, the grouping of the muscular lesions which corresponds roughly with the distribution of nerves, the course and progress of the disease, enter into the differential diagnosis.

8. *Atrophies following articular lesions.* History, localisation at diseased joints without tendency to become generalised, increase of tendon reflexes and other clinical features distinguish these from progressive muscular atrophy of myopathic origin. To distinguish them from myelopathies there is further the absence of R.D. and of fibrillary contractions.

CHAPTER II.

TREATMENT OF MUSCULAR ATROPHIES.

ALL who study the treatment of progressive atrophy in its literature and in actual practice, must be struck by the hopeless tone of the one and by the helpless inaction of the other. It is true that in the case of atrophies depending on the destruction of motor ganglion cells the latter once destroyed can never be restored; but there are other of these cells whose vitality is only impaired, not lost. Can these be strengthened by treatment to resist the morbid process, whilst at the same time the functional stimulation of the affected muscle is judiciously maintained? Drugs have been fully tried and for the most part found wanting. The same may be said of attempts to influence the disease through improvement in the general health. Climatic, hygienic, and hydro-therapeutic measures have proved useful, but not equal to the occasion. Therefore goes forth the verdict—"treatment is almost powerless," with the rider "try electricity." Now what may reasonably be expected of electricity—that *dernier ressort* of the baffled physician? This question has been very often asked and very differently answered. Erb, although he has never seen recovery in a true typical progressive muscular atrophy, "has seen improvement, relief, retardation of symptoms, and sometimes an arrest of the affection in individual cases, especially amongst those who have come early under electrical treatment." According to Haddon "electricity is occasionally of service, though in many cases it entirely fails." Gowers considers that electricity "fails, as a rule, to produce alone any effect on the disease." Here there is a "sliding scale" of appreciation amounting to a wide difference at its extremities. Is it permissible to suggest that *methods* may modify results? This becomes the less improbable in view of the very crude procedures that are sometimes employed. Those who see in electricity only a muscle-contracting stimulus, who ignore its nutritional and sedative effects, who measure "by cells" instead of by definite electrical units, whose ideas of current strength have not got beyond "weak," "moderate" and "strong," who delegate "an intelligent nurse" to *dab on* an unmeasured current or tetanise with "the faradic shock," are more likely to accelerate than to retard the progress of a muscular atrophy by resorting to electrical applications.

In dealing with muscular atrophies, treatment must be directed, not only to the atrophy, but to the lesion, if there be one, of which the atrophy is the manifestation. For purposes of treatment therefore muscular atrophies arrange themselves as follows:—

I. Atrophies due to lesions of the cord—(*myelopathic*). (Including degenerations consecutive to cerebral disease).

II. Atrophies due to primary degeneration of muscles (*myopathic*).

III. Atrophies due to lesions of peripheral nerves (*neuropathic*).

I. MYELOPATHIES.

It is certain that an electric current can be made to traverse the cord, and it is almost certain that such a current can thereby modify circulation, nutrition, and the finer molecular changes of the tissues that lie in its path. It follows, therefore, that so far as the integrity of the cord is concerned, the condition of the latter may be expected to improve. In muscular atrophies, dependent on lesions of the anterior cornua, it will generally happen that although certain of the ganglion cells are destroyed beyond recovery, there are others whose vitality is only impaired, not lost, and it is easy to conceive of such, that they may be so stimulated and strengthened by electrical treatment (which can certainly reach them) as to be the better able to resist the invasion of the microbe or toxine, or other pathogenic agency that may be at work to threaten their vitality.

Now, if a current be localised through the atrophied muscle, what may be expected? Not only will the muscle itself be stimulated, but the sensory nerves traversing its substance will be excited also, and through them the centres in the cord may be indirectly influenced. With a similar end in view, ingoing impressions arising from the stimulation of special cutaneous areas* may be brought to bear upon special segments of the cord.

Independently of any localised applications, some form of *general electrisation* (general faradisation or the hydro-electric or electro-static bath) is often useful. By such means the metabolic exchanges are quickened and modified, and the general condition of the patient favourably influenced.

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* Experimentally demonstrated by Hodge. The treatment of such areas by sinapisms and hydro-therapeutic methods are familiar instances of this; and almost equally familiar is the "faradic brushing" of the nape of the neck and spine in neurasthenia, of the lower angle of the scapula in hepatic affections, of the epigastric region in stomach trouble.

It is proposed first to describe the technique of certain procedures which, modified to suit individual cases, present a definite method of dealing not only with any muscular atrophy, but with almost every morbid condition that is open to the influence of electrisation. Recorded as below, these electrical formulæ may appear unnecessarily complex, but if followed out in one or two instances to begin with, they will in the end contribute to clear ideas ; and a closer inspection will at once show that no detail therein noted can be overlooked in any electrical application that lays claim to definiteness and accuracy.*

LOCAL TREATMENT OF THE CORD.†

A current may be made to traverse the cord (*a*) in a longitudinal (*b*) in a transverse direction.

(*a*). Longitudinal treatment of the cord.

Current :—

Form, continuous.
Intensity, 10-20 ma.
Density,‡ ·4 ma. to sq. in. (·07 ma. to sq. cm.).
Duration, 8-12 minutes.

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* These formulæ do not imply that electricity can be administered by rule of thumb. The actual "dose" (intensity, density, duration) must be decided at the moment of application, according to the medical and electrical skill of the operator. Electricity cannot be "dispensed." It had better be dispensed *with*.

† Abbreviations: + Positive pole. — Negative pole. Current, "continuous," "induced," "combined" (continuous and induced), "sinusoidal"; ma. milliampère; sq. in., square inch; sq. cm., square centimetre. "Labile," the electrode moved slowly up and down over the part. "Stabile," remaining stationary.

‡ The "density" of a current is the relationship of the intensity of the

Electrodes :—

Dimensions + 4 × 6 inches — 4 × 6 inches.
10 × 15 cm. 10 × 15 cm.

Position
Polarity
Method
+ stabile nape of neck, then labile over spine; — stabile.

On each alternate day.

b. Transverse treatment of the cord.

Current :—

Form, continuous.

Intensity, 10-20 ma.

Density + ·37 ma. to sq. in. — ·15 ma. to sq. in.*

Duration, 8 to 12 minutes.

Electrodes :—

Dimensions + 1·5 × 18 in. — 7 × 9 in.

Position
Polarity
Method
+ on spine, stabile; — on sternum, stabile.

On alternate days.

LOCAL TREATMENT TO THE MUSCLES.

There are several methods available, and the selection will depend upon whether the nutritional and other effects of the current are aimed at, or only muscular contraction pure and simple (electrical gymnastics).

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current to the surface area of the electrode, and may be expressed by a fraction, the numerator of which is the intensity, and the denominator the surface area of the electrode. Thus in the above case $\frac{10}{24}$ = ·4 absolute density, *i.e.*, ·4 ma. to the sq. inch, or in centimetres $\frac{10}{150}$ = ·07 ma. to sq. cm.

* To convert inches into centimetres multiply by $\frac{33}{13}$. To convert centimetres into inches multiply by $\frac{13}{33}$.

FIRST METHOD.

Current :—

Form, "combined."

Intensity 6 ma. Coil current just sufficient to produce slight muscular contraction.

Density + ·25 ma. to sq. in.; — 1·5 ma. to sq. in.

Duration, 5 minutes.

Electrodes :—

Dimensions + 4 × 6; — 2 × 2.

Position
Polarity
Method
+ stabile over spine or upper part of limb; — labile and stabile over muscles.

On alternate days.

(The writer uses a small roller-electrode. In passing this over the muscles slight contractions are secured, and at the same time a useful form of mechanical pressure on the muscle).

SECOND METHOD.

Place the indifferent electrode on a remote part of the body. The other electrode (of testing-size and having an interrupting handle) is placed over the nerve where the latter is superficial, or over the motor point of the muscle. Then by means of suitably timed interruptions, rhythmical contractions may be induced in individual muscles, or in groups of muscles, either current being used, or both combined.

THIRD METHOD.

Using the current of the induction coil, the two electrodes held near each other are drawn slowly backwards and forwards over the affected muscles. This is usually what is meant by "electricity" in the crude electrical procedure of the "Weir-Mitchell system."

It is well to remember in the electrisation of diseased muscles; (1) that strong currents and powerful contractions are to be avoided, for the obvious reason that they are calculated to exhaust the excitability of a weakened muscle; (2) that muscular fibre ought to be in a slightly relaxed position during electrisation; (3) that it is well to extend treatment to such sound muscles as are likely soon to be attacked.

III. PERIPHERAL TREATMENT.

There is some experimental and much clinical evidence to show that the circulation of the cord and the nerve cells of its grey matter,* can be influenced by peripheral excitation. Perhaps every internal organ has its corresponding skin area. In the case of spinal cord diseases this form of excitation is most easily effected by faradocutaneous brushing. The electric douche is highly effective for the same purpose; but its technique involves much detail and cannot be entered into now.†

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* And probably the condition of every structure and organ of the body.

† "Hydro-Electric Methods in Medicine." (H. K. Lewis, London).

The treatment of "tender points" may be referred to here. An electrode varying in size with that of the painful area is attached to the positive pole and allowed to remain stabile on the tender point for 5 to 30 minutes. The current density for such a purpose ought not to exceed, for short applications, 1 ma. per sq. inch, and in longer ones may even be so low as ·2.

4. *General Electrisation.*—The methods of general electisation available in muscular atrophies are static charging, the hydro-electric bath (with continuous, induced, or sinusoidal currents), high frequency, high potential currents, and the method known as "general faradisation." Of these the last is the only one likely to be within the ordinary resources of the practitioner. It is carried out by placing the patient's feet upon a flat metal plate (kept warm if necessary) attached to the negative pole of an induction coil. The other pole is attached to a circular electrode (3 inches in diameter) furnished with a handle and having a slightly convex surface. This is used "labile" over the various parts of the body in succession. Commencing with the neck and using a suitable current, the electrode is moved slowly up and down each side of the cervical spine (3 minutes), and a few times up and down the anterior border of each sterno-mastoid. The current is then strengthened until capable of producing muscular contraction or just short of this, and the whole length of the spine below the cervical vertebræ is treated in a similar way (4 minutes). The breast, abdomen, and extremities are then dealt with (about 5 minutes) the whole proceeding occupying about 12-15 minutes. But special time and attention may be devoted to special parts and special symptoms, *e.g.*, to the abdomen in obstinate

constipation. When it is desired to pass a current through the head, this may be done by applying one hand (wetted) to the patient's forehead, and with the other hand gradually taking hold of the positive electrode; the current passes *viâ* the body of the operator from the head to the foot of the patient. The looser or firmer hold of the electrode regulates the strength of the current. The duration of the head application may be one minute, and the entire procedure of general faradisation should never exceed 15 minutes.

5. *Subaural Galvanisation.*—An attempt is often made (by acting on vaso-motor pathways through the sympathetic ?) to influence in a special way the circulation and nutrition of the cord. It is carried out by placing one electrode (generally the negative) 2 × 2 (5 × 5 cm.) "at the angle of the jaw against the hyoid bone, with the surface directed backwards and upwards towards the vertebral column," stabile. The other pole 4·5 × 2 (5 × 12 cm.) is used stabile, and sometimes labile on the opposite side of the neck at the level of 5-7 cervical vertebræ. The intensity may be 2-5 ma., the duration 1-3 minutes, and if considered advisable the application may be made to both sides. Not only in progressive muscular atrophies, but in various paralyses, spasms, migraine, &c., excellent results are attributable to this method, as all who have systematically used it are able to affirm. How far such results are attributable to the sympathetic, is another question; a current following the path indicated must strike many other important structures, such as medulla, cervical part of cord, and pneumogastric.

By means of the foregoing measures, or selections or slight modifications of them, it will be found possible to deal with muscular atrophies of every class. Some of the commoner cases which are likely to present themselves for treatment may be briefly considered.

CHAPTER III.

APPLICATION OF FOREGOING FORMULÆ.

I. MYELOPATHIES.

1. *Progressive Muscular Atrophy (Myelopathic).*—Local treatment to cord and muscles as detailed above. General electrisation and subaural galvanisation are often indicated in addition.

2. *Amyotrophic Lateral Sclerosis.*—Treatment on the same principles as progressive muscular atrophy, modifications to suit local symptoms.

3. *Atrophy from Myelitis.*—Deal with the myelitis by treatment directed chiefly to the spine, using only mild currents (3-4 ma. at first, later 8-10 ma.), and of short duration (1-5 minutes). Positive pole stabile to tender points. In myelitis from compression (kyphosis) place the electrodes immediately above and below the curvature, using currents as above (Erb).

4. *Atrophy in descending secondary degenerations in "cerebral disease."*—Try long continued action of the positive pole (5 to 8 ma.) with electrodes over the cord stabile; with subaural galvanisation.

5. *Atrophy in Locomotor Ataxy.*—Tabes is sometimes accompanied by a very rapid wasting of the muscles, but this atrophy being only a condition superadded to the primary and distinctive symptom, an "epiphenomenon," its treatment will be that of the primary disease, and will follow the lines already sketched out in general

methods. But it will be well to use shorter applications (3-5 minutes), and milder currents (8-10 ma.); adding local treatment directed to the muscles. Special symptomatic treatment, such as the stabile action of the positive pole over the roots of painful nerves and over spots tender on pressure, may also be advisable. Farado-cutaneous brushing of the skin of the back and limbs (method of Rumpf) is an expedient always to be remembered. The hydro-electric bath is, according to the present writer's experience, a most useful measure if medically administered.

Infantile Paralysis.—Treatment must be commenced early and carried on for months—even for a year with occasional breaks. Here a caution may be added against much preliminary "testing." It is to be remembered that cutaneous sensibility is generally unimpaired, and a very young child once frightened by a strong current, will on subsequent occasions scream at the very sight of the battery or the bath. There are several methods of treatment.

1. When the lower limbs are affected it is a good plan to take a small wooden or earthenware vessel (*e.g.*, a footbath) containing water at about 98° F., and having hung an electrode at each end, place the child sitting in the bath, with some light covering over the shoulders. Commencing at zero the current is gradually increased after the child is in position. The application may last 5-10 minutes, and be repeated 3 or 4 times a week, or even daily.*

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* The current from street mains must not be used with any improvised bath of this sort. Such currents are safe only with an efficiently insulated bath.

Those who see in the existence of R.D. a reason for using a continuous current only will select a suitable intensity (10-15 ma. or less), taking care that the electrodes do not touch the child's body.

2. Another, and probably a better method than either of these, is to place the foot or hand in a basin of warm water, at 98° F., containing the negative electrode. The positive electrode, 24 sq. in. (150 sq. cm.), is over the cord or upper part of the affected limb ; 8-10 ma. is used for 8-10 minutes, 3 times a week. A "combined" current is suitable here. Some interruptions may be made to produce muscular contraction ; but these electrical gymnastics must be neither violent nor prolonged.

II. MYOPATHIES.

In the absence of anything like a satisfactory pathology, the methods of general electrisation offer themselves as the most suitable procedures. The use of the sinusoidal currents from the street mains, applied through the water bath, is an excellent way of dealing with these conditions (Gautier and Larat). But whatever form general electrisation may take, there must also be local treatment of the muscles according to one or other of the ways detailed in general methods. Erb (1887) recommends a treatment similar to that used in myelopathic atrophies, inasmuch as, notwithstanding the negative condition of the cord, he considers it by no means disproved that "we have not to do with a trophoneurosis of central origin."

III. NEUROPATHIES.

1. *Peripheral Neuritis.*—In accordance with traditional methods, and with the physiological fact that the positive pole possesses a sedative action and the negative a stimulating one, it is usual to trust at first to the stabile action of the anode. Those who see in the phenomena of electrotonus a satisfactory foundation for a theory of electro-therapeutics need not be disturbed in their simple faith. But there are others who, apart from the question whether a sedative rather than a stimulant action is indicated in this degeneration, may try to think out the course and action of an electric current, as as in obedience to the law of Ohm, and the laws of electrolytic action, and cataphoresis, and the establishment of actual and virtual poles in its path, it traverses the unexposed nerve of a living body. To such it may seem necessary to ask whether it is easy, or even possible, to set up at will and to localise these physiological conditions of electrotonus. Tempting as it is to seize upon a principle so easy to apply, it will probably prove necessary to fall back upon an enlightened empiricism. Still it is difficult to do better than to try at first the stabile action of the positive pole, the other being placed upon an indifferent part of the body, or preferably (according to Erb) on a more central part of the diseased nerve, or upon the corresponding segment of the cord. The intensity may be 2-8 ma.; the duration 2-10 minutes; the size of the electrode 4 × 6 inches. When degeneration is further advanced the use of the negative pole alternately with the positive is recommended. If peripheral

neuritis be regarded as a degeneration of nerve cells accompanied only sometimes by inflammation, and that inflammation affecting structures other than the nerve tissue itself, the above treatment may appear to err on the side of over cautiousness. Individual pathological views must necessarily influence practice. In the case of extensively distributed neuritis, the writer has constantly found the hydro-electric bath of service, combined with the local treatment of tender points when present.

2. *Atrophies following injury of individual nerves.*—There are two forms of atrophy from compression, (*a*) a slight form, (*b*) a severe form. In the latter case, the severity of the injury produces histological changes, neuritis and degenerative atrophy. In the slight form, the injury is perhaps more of an "irritation"; the reactions are qualitatively normal, and the atrophy is known in the language of electro-diagnosis as "simple" atrophy. In the severe form there is R.D. Apart from the severity of the injury, there exists doubtless, in many cases, a predisposition on the part of the patient which acts as the determining factor in producing a degenerative, rather than a simple atrophy.

In slight traumatic lesions such as sleep paralyses, &c., causing only slight changes in the nerve fibres, the direct action of the negative pole with a weak current is usually an effective treatment, especially if this be combined with stimulation of the affected muscles. In degenerative atrophy (and R.D.) following section and severe lesions of nerves it has usually been considered that some continuity must have been restored before much can be expected from electrical treatment, but there is evidence to show that the latter applied to the seat of lesion, is not without influence on the process of repair, and that it may do something more than keep the muscles exercised pending the regeneration of the nerve. A recent experiment of Friedlander (of Wiesbaden) is as follows:—He cut the sciatic nerves of a dog. Daily galvanisation was carried out on the left side, not on the right. At the end of a month the left leg had recovered, and excepting that it became more easily fatigued it appeared as sound as the front legs. The right on the contrary was still paralysed, there was ulceration of the foot and atrophy of the muscles. He concludes that in traumatic paralysis the regeneration of nerve trunks is hastened by electrisation, and that if employed early it may prevent the occurrence of muscular atrophy.*

Brachial plexus—Erb's paralysis. (5th and 6th nerve trunks). (Fall or blow, or other injury to the neck. Forcible traction. Cold. Injury during operations).

Place one electrode 2 × 2 inches over the lower cervical vertebræ, the other, of rather larger size in the axilla; use a current of 5 ma. for 5 minutes. Then by means of labile applications to the atrophied muscles (faradisation if they react to this) according to one or other of the procedures in general methods, induce slight muscular contractions.

Circumflex nerve.—Its injury results in paralysis and atrophy of deltoid. (Pressure of crutches, falls on shoulder, exposure to cold, rheumatism, dislocations).

Treatment.—5 ma., stabile for 5 minutes through the muscle; then stimulation by labile applications or interruptions or faradisation (see general methods). Avoid powerful and exhausting contractions. Support the arm

* "Rev. Int. d'Electrotherapie," 1897.

horizontally, on the principle of always keeping the muscle relaxed during electrisation.

Musculo-spiral.—Oftener than any other, this nerve is paralysed singly. Owing to its course it is liable to injury from fracture of humerus, dislocation, callus, pressure from falling asleep in awkward positions, &c.

Supplying as it does triceps, muscles on back of forearm, integument of same parts, radial side of back of hand, radial side and ball of thumb, ulnar side of thumb, index finger, middle finger, and half of ring finger, it forms by its wrist drop, limitation to one side, &c., a characteristic paralysis and atrophy, and the incidence of the paralysis on particular muscles usually enables the injury to be accurately localised by electrical and other testing.

Treatment as for traumatic paralysis of individual nerves. Continuous current negative pole, 3 to 5 ma., for 5 minutes over seat of injury, one electrode at front, the other at back of arm. All the affected muscles are then made to contract moderately, and with distinct intervals, by one or other of general methods for 3 or 4 minutes. Duration of entire application not more than 10 minutes. Every day or every other day.

Ulnar nerve.—Its superficial position at elbow and wrist render it liable to separate injury (pressure, section, neuritis, &c.). Supplying as it does flexor carpi ulnaris, the hypothenar muscles, adductor pollicis, inner head of flexor brevis, all the interossei the two inner lumbricales, and conjointly flexor p. digitorum, all of these may be paralysed or atrophied, involving not only loss of appropriate physiological action, but a characteristic deformity (*main en griffe*), due to paralysis of the interossei and the action of their antagonistic muscles (see Chap. X.). Treatment to seat of injury as in last case, followed by stimulation of the affected muscles. Altogether 10 minutes. If there be anæsthesia, stimulation by faradic brush from the first.

Median nerve.—When the median nerve alone requires treatment it is generally the result of wounds. In such a case the atrophy falls upon the pronator muscles—pronator rad. teres and pronator quadratus, the flexor carpi radialis, flexor sublimis digitorum, part of flexor profundus digitorum, and thenar muscles excepting adductor pollicis and inner head of flexor brevis pollicis.

Treatment to the nerve and muscles as in the case of the ulnar nerve.

Sciatic nerve.—(Pressure, neuritis, rheumatism, dislocation, &c.).

Taking electrodes 4 × 6, place one over the lumbosacral region, and the other below the sacro-sciatic foramen. Let a continuous current (10 to 25 ma.) be used daily for 5 minutes, afterwards exercising the muscles by unipolar or other form of stimulation, as detailed in general methods. If sciatica be present, this must be treated, first, by means of electrodes 4 × 6, one being placed over the sacrum or lumbar vertebræ, the other along different points of the nerve, especially over tender points (10 to 20 ma., 5 to 10 minutes). Faradisation by strong currents or peripheral excitation by the faradic brush, have also been of service here. The atrophy will be dealt with by one or other of the general methods of muscular stimulation.

Peroneal nerve.—(Pressure, neuritis, falls, &c.). Tib. ant., Ext. l. dig., Peronei, Ext. b. dig. affected. There is foot-drop, loss of dorsal flexion of foot, and of extension of toes. Talipes equinus results ultimately. An electrode 4 × 4 is placed at the outer part of popliteal space near the origin of the nerve, and the other, a larger one, a few inches above patella. Stabile application, 5 to 15 ma., for 5 minutes. Then stimulation of individual muscles just sufficient to produce their contraction.

Internal popliteal.—Flex. l. dig., Tib. post., Popliteus, calf muscles, muscles of sole, affected. Plantar flexion of foot, adduction of foot, plantar flexion of toes lost. Talipes calcaneus eventually. Treatment as in the case of the peroneal, but the lower electrode will be placed over the great transverse fold of the popliteal space, and the muscles requiring treatment will be those just enumerated. In all the above cases where there is anæsthesia, peripheral stimulation by the faradic brush or faradic water douche will form an important part of the treatment, and prove beneficial not only to the anæsthesia, but also to the other symptoms of the case.

3. *Atrophies consequent on Articular Lesions.*—It is well known that following in the wake of hip disease, fractures, diseases of the knee joint, &c., there often occur muscular atrophy, paralysis, and contracture; and of these the most frequent, as well as usually the first to appear is atrophy. This is not in consequence of mere fixation and disuse. Charcot has shown it to be a "reflex" effect; something of the nature of an inhibition exercised upon the cord by the afferent nerve fibres which are irritated at their articular distribution. There is not R.D. nor fibrillary contractions, neither is there any tendency to the atrophy becoming general. More than one joint may be affected, that is to say, there may

be "multiple" manifestations, but the atrophy is not "progressive."

If the cause be no longer active, that is to say, if the joint disease be cured, this form of atrophy presents few difficulties in treatment, and almost invariably yields to patience and well directed effort. Electrical treatment consists in localised applications to the muscles as already explained, the roller electrode playing a most useful part.

Not specifically mentioned in the foregoing classification there is a form of paralysis and contracture following fracture of the forearm described by Volkman,* and termed by him "ischæmic." It is stated that the electrical reactions are not altered, and the condition has been considered due to "venous stasis, probably from pressure of narrow splints, and insufficiently padded bandages especially in the neighbourhood of the wrist," which produce an interstitial inflammation. Owing to "inflammatory fibrosis of the muscles" treatment is unsatisfactory; but it should consist, in a recent case, of forcible stretching under an anæsthetic, of localised electrisation according to one of the foregoing methods, of manipulations and passive movements, and eventually, perhaps, tenotomy. When ischæmic claw-hand has existed for sometime cure can scarcely be hoped for. It is not, as was formerly thought, pressure on the nerve which causes the paralysis leading to contracture. The latter is due to "ischæmic muscular shortening." A paralytic contracture would be slower in its onset. The contracture in question may establish itself in a few days.

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* "Centrablatt für Chirurgie," 1881, no. 51.

CHAPTER IV.

I. THERAPEUTIC EXERCISES.

THERAPEUTIC exercises and passive manipulations are in the highest degree helpful, but they must be judicious, and in the best sense of the term *scientific*. As an example, consider a condition just dealt with, viz., atrophy consecutive to an articular lesion. It is evident that inasmuch as movement and manipulation are in certain stages the very antithesis of the treatment suitable for joint trouble, such measures must be most carefully entered upon, and any remaining or returning signs of disease in the articulation must be anxiously looked for. Manipulations by persons other than medical men are in such cases quite out of place, not only because the former have no adequate knowledge of the rationale and risks of the procedure, but because they have not eyes to see a threatened recrudescence of the articular mischief.

Movements and manipulations are to be employed in muscular atrophy, not only with a view to their direct effect on the condition of the muscle itself, but for their beneficial influence on circulation, respiration, and other functions. The writer, therefore, ventures to reproduce the following from his essays on physical therapeutics*:—

In the year 1776, at Ljunga, in Sweden, was born Pehr Henrik Ling. The thirteenth year of the present century found him, after many difficulties and much discouragement, at the Royal Central Institute of Stockholm, under Government Patronage and State aid, teaching and practising a system of gymnastics of a novel and peculiar kind. Built up upon more or less fantastic theories,* the greater part of Ling's system has long since perished, but one of its great distinguishing features still survives. This is the "combined" or "resisted" movement. Here two forces participate—one "makes" the movement, the other "resists" it. For example, the person being exercised is told to "bend the forearm until the fingers touch the shoulder," the other person with carefully judged force "opposes." This principle of combining patient and "operator" into one person to carry out a movement was quite new. It is true that long before this—so far back as 1573—Mercurialis had published a book, "De arte Gymnastica," in which movements are described as "active," "passive," and "mixed"; but the latter are altogether different from the "combined" movements of Ling, the essential feature of which is the combination of movement with resistance. It is these movements which have given name and fame to the so-called "Swedish movement cure," and which constitute the basis of many modern systems of "cure gymnastics." The therapeutic movements of Schott are the resistive movements of Ling adapted to the treatment of chronic heart disease.

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* "Treatment," May, 13, 1897.

The medical man who proposes to carry out resistance movements himself will have no difficulty in devising methods. No elaborate "technique" is necessary. The patient is directed to perform a certain movement, and as the physician himself "opposes" it, it is evident that he has control of the exact amount of work his patient is called upon to perform. The chief articulations of the extremities are taken in succession and put through the various movements of which they are capable, a judicious resistance being always opposed. The trunk also is flexed and extended, and bent laterally to either side, and rotated in each direction, always against resistance. During the whole time of the exercise the patient's method of breathing is carefully watched, and if necessary the instinctive tendency to hold the breath is obviated by the usual device of making him "count." The form of exercise, its duration, and the amount of resistance is adapted to the case under treatment. When directed against chronic heart disease* it is not intended that the respiration be materially accelerated; any approach to fatigue is to be avoided, and each movement is to be carried out only once, followed by a distinct interval of rest (Thorne). The resistance must be such as is easily overcome. What is known in the physiology of exercise as "effort" always hinders for the moment respiration and circulation.

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* Ling regarded life as the blending together of three elements—the dynamic, the chemical, and mechanical, "which through their opposite action produce variety in vital phenomena" (Busch).

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* Stenosis of mitral or aortic orifice or both. Incompetence of either or both. Dilatation. Weakened heart. Angina pectoris apparently both of neurotic and of organic causation. Aneurism of the heart or great vessels (Thorne). Resistance gymnastics cannot be allowed in cases of universal arterio-sclerosis of high degree, the increased energy of the heart being only compensated in an incomplete and unsatisfactory manner by corresponding dilatation of the arteries. . . . . It is a matter of course that patients who have once had apoplexy are not to be treated by resistance gymnastics, nor those who suffer from aneurism. [Groedel (of Nauheim), "Lancet," April 17th, 1897].

When the medical man delegates an assistant to carry out these exercises it is necessary (1) that the assistant be instructed by actual demonstration; (2) that he be furnished with written rules and illustrations of each exercise; (3) that before being intrusted with a case he have the exercise suitable for that individual case "administered" to him by the prescriber (in order to be able to gauge by his own feeling the exact amount of resistance force to be used); (4) that especially he be made familiar with those signs and symptoms in the patient which are to be accepted as signals of *cease exercise.*[*] The Ling movements selected by Schott are detailed by Dr. Thorne.[†] Although devised for chronic heart affections these movements have really a much wider range of application than this, and furnish a convenient "formula" for dealing with a large variety of morbid conditions. It may be useful therefore to state that in point of number and sequence the exercises are as follows:—

1. *Commencing Position:* Arms extended in front of body on level with shoulder; palms in apposition. *Exercise:* Carry arms outwards until in a line with each other. Resistance to movement. Back to original position. Resistance.

2. *C.P.:* Arms hanging, palms forwards. *E.:* Flex one forearm until fingers touch shoulder. Resistance. Back to original position. Resistance. Repeat with other arm.

3. *C.P.:* Both arms hanging, palms forwards. *E.:* Raise arms outwards until thumbs meet over head.

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[*] Dilatation of alæ nasi, drawing of the corners of the mouth, duskiness and palior of the cheeks and lips, yawning, sweating, palpitation (Thorne).

[†] "The Schott Methods." W. Bezley Thorne, M.D. (Churchill).

Back to original position. Resistance to both movements.

4. *C.P.:* Hands pressed together in front of lower part of abdomen, fingers being flexed in such a way that the dorsal aspect of each second phalanx is in apposition with the same aspect of the corresponding phalanx of hand of opposite side. Thumbs kept extended. *E.:* Raise arms until hands are on level with vertex. Back to original position. Resistance both ways.

5. *C.P.:* Arms hanging with palms against thighs. *E.:* Raise arms in parallel planes until vertically extended. Back to original position. Resistance to both movements.

6. *C.P.:* Body erect, knees straight. *E.:* Flex trunk. Resistance with hand over upper part of sternum. Back to original position. Resistance with hand at back of neck.

7. *C.P.:* Body erect, face forwards. *E.:* Rotate trunk (without moving feet) as far as it can be carried to one side. Resistance. Rotate as far as possible to the other side, and back to original position. Resistance.

8. *C.P.:* Body erect, face forwards. *E.:* Flex trunk laterally. Resistance. Then completely over to the other side and back to commencing position. Resistance.

9. *C.P.* and *E.:* As in No. 1, but with fists clenched.

10. *C.P.* and *E:* As in No. 2, but with fists clenched.

11. *C.P.:* Arm hanging down. *E.:* Complete circumduction of extended arm in largest possible circle travelling forwards in ascent, backwards in descent. Resistance throughout.

12. *C.P.:* Arm hanging. *E.:* Arms moved backwards and upwards as far as possible without bending trunk

forwards. Back to commencing position. Resistance in both directions.

13. *C.P.:* Standing with one hand resting on chair or table. *E.:* Flex thigh of opposite side on trunk, leg hanging downwards from knee. Resistance by hand placed immediately above knee. Back to *C.P.* Resistance by hand below thigh or sole of foot.

14. *C.P.:* As in 13. *E.:* The extended leg is bent backwards and forwards at the hip, and as far as it will go in either direction. Resistance at ankle.

15. *C.P.:* Standing with hands resting on chair or table. *E.:* One leg is flexed on thigh. Resistance at heel. Back to *C.P.* Resistance at instep.

16. *C.P.:* Standing. One hand on chair. *E.:* Leg of opposite side extended at the knee is raised outwards from hip. Back to *C.P.* Resist at ankle.

17. *C.P.:* Arms extended horizontally outwards. *E.:* Rotate from shoulder joint. Resistance by closing thumb and forefinger in a ring round wrist.

18. *C.P.:* Operator supports wrist. *E.:* Extend hand on forearm. Resistance at dorsal aspect of metacarpo-phalangeal articulation. Flex hand on forearm. Resist on palmar aspect.

19. *C.P.:* Sitting or standing. *E.:* Flex and extend feet to extreme limits. Resistance on dorsal and plantar surfaces respectively.

II. Manipulations.

Of these the most useful are the following:—

1. *Stroking movements* with the flat of the hand over the surface of the skin, with various degrees of pressure, in a

direction from the extremities to the trunk, and as far as possible in the line of the muscular fibres.* The effect of this is (1) to produce an excitation of the skin and to hasten circulation in the vessels; (2) to increase the activity of osmotic changes between capillaries and tissues; (3) to stimulate the lymphatics and make them take up extravasations and exudations, and (4) mechanically to drive all the fluids onward and away from the diseased part.

2. *Friction movements.*—Here both hands must work together, but not with the same kind of movement. One moves in a circular or elliptical manner and with considerable pressure, mainly exercised with the ends of the fingers, while the other follows with a stroking, pushing motion, the idea being that one "disaggregates" the morbid products, *i.e.*, separates and perhaps breaks them down, the other forces them on into the circulation, or at least distributes them over a larger area, thereby making absorption more easy.

3. *Kneading movements.*—A fold of tissue is grasped by the hands, and a squeezing, rolling, twisting movement is imparted, one hand following the other and "taking over from it," so to speak, the fold of tissue as the other gives it up. Both hands travel steadily from the extremities towards the cardiac centre. Without actual demonstration it is not possible to convey any adequate idea of this movement, but its object is clear. A muscle in action is something like a beating heart; by every contraction it forces out a volume of blood, and in relaxation fills again, and so the circulation is helped on. The alternate pres-

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* If the space be too small for the hand these movements are done with one or more fingers.

sures and relaxations of the above manipulation by emptying the muscles (like squeezing a sponge), and letting them fill again, are so far equivalent to the contraction and relaxation, the emptying and filling of the active muscle, in fact they mechanically reproduce one effect of exercise. Like a muscle in action this manipulation helps forward the circulation, and this as well as the "derivation" of blood from internal organs to the muscles relieve congestions in the former. The benefit of exercise is thus secured without such an expenditure of energy as is involved in the ordinary forms of active exercise. The "tone" of a muscle is also improved by this manipulation, and flabby and exhausted muscles are found to become redder and firmer.

The pathology of fatigue is obscure, but it is not improbable that the sensation of fatigue is intimately associated with chemical changes that occur in working muscles. A sense of weariness is felt after injection of a dilute solution of phosphoric acid, or dissolved extract of meat. "Muscle is fatigued far more rapidly than nerve, and the fatigue begins in the muscle and not in the nerve. It seems to be the weakest link in the chain between nerve and muscle which is affected during excessive action, viz., the motor end-plate."* By increasing the activity of the circulation waste products are removed or altered; the latter result is thus secured by the above manipulation. Further, it may be that some so-called functional nervous disorders consist in a derangement of the molecular elements of the structure; therefore it seems probable enough that such stretchings and twistings as those described may alter this morbid condition and bring about a normal rearrangement of the ultimate anatomical elements, in some such way, perhaps, as does the surgical proceeding of nerve stretching.

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* Landois and Sterling, "Textbook of Physiology," p. 615, vol. ii.

Reviewing broadly the effects of the foregoing manipulations it appears that their tendency is to promote the circulation of blood and lymph, to produce the breaking up and absorption of morbid products, to stimulate contraction of muscular fibre, to excite nerve action, to elevate local temperature. With discrimination, therefore, such procedures may be called in to influence the processes of circulation, respiration and assimilation. They may be useful in conditions of general debility, in non-acute rheumatic and post-rheumatic affections, in articular and peri-articular troubles, in chronic muscular affections, whether the latter depend on faulty innervation, or are of so-called idiopathic origin.

When delegated to an assistant it is necessary, as in the case of the foregoing exercises, that the particular manipulation be demonstrated to him, and full instructions given as to its duration, frequency, and general object.

III. DRUGS.

There are certain drugs whose aid may be usefully sought. So far as the writer's experience goes these are about five in number:—(1) Strychnia by mouth and intramuscular injection; (2) Potassium iodide in myelopathies of old standing, or specific origin; (3) Ergot; (4) Mercurial treatment in spinal cord diseases of syphilitic origin; (5) Arsenic. Of these by far the most important is strychnia. Its subcutaneous injection in "chronic muscular atrophy" is highly thought of by Gowers.* "In seven almost consecutive cases in middle life, this treatment has been followed by arrest within a month of its commencement, and the arrest has been permanent in all cases but one." Results so brilliant as this are not probably within the experience of many, either with this or any other form of treatment. It may here be mentioned that the usual textbook formula that "strychnia and electricity are contra-indicated in lateral sclerosis" is true of strychnia, but not of every form of electricity.

IV. HEAT.

The application of heat in various forms to the cord or atrophied muscles has been recommended. The writer has availed himself of his radiant heat apparatus for experimental work in this direction, but his observations have so far been too limited to base conclusions upon.

V. COUNTER-IRRITATION.

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INDEX.

Abnormal contraction, laws of, 22
Amyotrophic lateral sclerosis, treatment of, 106
Ankle clonus, 80
—— jerk, 80
Ataxy, 81
Atrophy from cerebral disease, 106
—— injury of brachial plexus, 111
—— —— circumflex nerve, 111
—— —— consequent on articular lesions, 114
—— —— individual nerves, 110
—— —— int. popliteal, 114
—— —— median nerve, 113
—— —— musculo-spiral nerve, 112
—— —— peroneal nerve, 113
—— —— sciatic nerve, 113
—— —— ulnar nerve, 112
—— myelitis, treatment of, 106
—— in infantile paralysis, 107
—— ischæmic paralysis, 114
—— locomotor ataxy, 106
—— myopathic, 3

Conjunctival reflex, 79
Contraction of iris reflex, 79
Counter irritation, treatment of atrophy by, 125
Current, direction of, 17
Cutaneous reflex, 79

Deep reflex, 79
Density, 16
Drugs, treatment of muscular atrophy by, 124

Elbow jerk, 81
Electrical exploration by constant current, 8
—— —— coil currents, 11
—— —— mixed effects, 21
—— —— sources of error in, 19
—— —— technique of, 7
—— reactions, 7
—— —— form of report for, 37
Electro-cutaneous sensibility, 54
—— —— diagrams of, 59
—— —— examination of, 55
—— —— of various parts of body, 56
Electro-diagnosis, instrumentation for, 5
—— limitations of usefulness of, 35
—— prognosis by, 36
—— usefulness of, 4
Electro-muscular contractility, laws of, 7
Electrotonus, 20
Excitability, normal, 24
—— —— course of changes in, 26
—— —— diminished, 25
—— —— increased, 25

Fatigue, pathology of, 123
Front tap contraction, 80

Heat, treatment of muscular atrophy by, 125

Inco-ordination, 81

Knee jerk, 79
—— how to elicit, 80
—— physiology of, 80

Manipulations, 121
—— Passive, 116
Motor points, 18, 39
Movements, cases useful in, 124
—— friction, 122
—— indications for, 124
—— kneading, 122
—— stroking, 121
Muscles, action of, 65
Muscular atrophy, classification of, 90
—— —— —— for treatment, 97
—— —— local treatment of cord in, 99
—— —— symptoms of, 91
—— —— treatment of, 96
—— —— —— various forms of, 106
Muscular sensibility, 77
Myelopathies, 97
—— general electrisation in, 98-103
—— local treatment to muscles in, 100
—— peripheral treatment in, 102
—— subaural galvanisation in, 104
—— treatment in, 126

Neuropathies, treatment of, 109

Paralysis, definition of, 77
—— ischæmic, 114
Peripheral neuritis, treatment of, 109
Progressive muscular atrophy, myelopathic, 106
—— —— —— treatment of, 106

Quantitative changes, 23-25

Reaction of degeneration, anomalous forms of, 30
—— —— diagnostic value of, 34
—— —— formula of, 27
—— —— partial, 30
—— —— theory of, 31
—— —— typical form of, 29
—— of exhaustion, 21
—— normal formula of, 8-26
Reflex action, 78
—— arc, 79
—— centre, 78
—— cutaneous, 79
—— muscle, 79
Resistance of body, 19
—— —— in diagnosis, 57

Sensory nerves, topography of, 59
Spinal cord, 81
—— —— cervical enlargement of, 82
—— —— extent of, 81
—— —— focal diseases of, 85
—— —— lumbo-sacral enlargment of, 82
—— —— nerve pairs, 82
—— —— plates of anatomy and pathology of, 82-85
Superficial reflex, 79
System diseases, 83

Tendon reflex, 79
Therapeutic exercises, 116
—— —— movements, 116
—— —— of Schott, 119

Wrist jerk, 81

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